EXHIBIT C18

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY

IN RE: JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES AND PRODUCTS LIABILITY LITIGATION ***THIS DOCUMENT RELATES TO ALL CASES***	**MDL NO. 16-2738 (FLW) (LHG)**

EXPERT REPORT OF GREGORY DIETTE, MD, MHS FOR GENERAL CAUSATION *DAUBERT* HEARING

Date: February 25, 2019

Gregory Diette, M.D., M.H.S.

I. SCOPE OF REPORT

I was retained by Johnson & Johnson and Johnson & Johnson Consumer Inc. to review the epidemiological literature regarding the hypothesized connection between talc or asbestos in talc and the development of ovarian cancer.

II. MY QUALIFICATIONS

I am a professor of medicine at the Johns Hopkins University School of Medicine. I hold joint appointments in the Departments of Environmental Health Sciences and Epidemiology in the Johns Hopkins Bloomberg School of Public Health.

I received my M.D. from the Temple University School of Medicine. I completed my residency at the Hospital of the University of Pennsylvania and performed a fellowship in pulmonary and critical care medicine at Johns Hopkins. I received my M.H.S. in Epidemiology and Clinical Epidemiology from the Johns Hopkins Bloomberg School of Public Health. Currently, I am an attending Physician at the Johns Hopkins Hospital and the Johns Hopkins Bayview Medical Center, practicing both inpatient and outpatient care.

My areas of clinical expertise include internal medicine, pulmonary medicine and critical care medicine. My areas of research include environmental impacts on lung disease and epidemiology of chronic diseases. I have published more than 200 studies in peer-reviewed journals on a variety of medical and scientific subjects, including the epidemiological study of disease causation, disease risk factors and gene expression, as well as the health effects of environmental pollutants. In addition, I am a peer reviewer for a number of journals. I have also repeatedly lectured and instructed on advanced research methods in epidemiology.

I currently hold multiple positions related to teaching and clinical research. I am an attending physician at Johns Hopkins and a member of the American Thoracic Society, where I served on the Board of Directors and have participated in a number of its teaching programs, including the Methods in Epidemiologic, Clinical and Operations Research program. I also previously served as the Director of Clinical Research in the Division of Pulmonary and Critical Care Medicine for almost 14 years.

Additional information pertaining to my background and qualifications can be ascertained from my curriculum vitae, which is attached to this report, together with other required disclosures. I am being compensated at a rate of $485 per hour for my work on this case and $600 per hour for testimony.

III. SUMMARY OF OPINIONS

The body of relevant epidemiological evidence does not support a causal connection between perineal use of talcum powder products (whatever constituents those products may contain in addition to talc) and ovarian cancer. As fully set forth below:

1. The epidemiological literature shows a non-existent association or, at most, a small association between perineal talc use and ovarian cancer that constitutes only weak epidemiological evidence. Because any purported association demonstrated in the literature is weak, it may well be attributed to factors such as confounding, bias or chance.

2. Studies have not consistently shown an association. The prospective epidemiological studies (cohort studies) do not show a statistically significant association; the hospital-based case-control studies do not show a statistically significant association; and only a subset of the population-based case-control studies show a statistically significant association. If consistency could be drawn from these inconsistent results, it would be a consistency of null results because case-control studies, which are more easily subject to certain biases and confounding factors, are not the best evidence for proving causation.

3. Evidence of a dose-response relationship is lacking. None of the cohort studies reveals a dose-response relationship, and only a handful of case-control studies, including those analyzing "cumulative" talc use, have purported to find one. Moreover, study authors and plaintiffs' experts all agree that there are major challenges to interpreting the study findings on dose-response because there can be no assurance that any estimates of talc use are accurate or valid. Indeed, there is not a single epidemiologic study of ovarian cancer and talcum powder that has used, or purports to have used, a validated measure of talcum powder use. Without a validated measure of talcum powder use, it is impossible to correctly determine whether or not an exposure occurred or the quantity of purported exposure, casting considerable doubt on any purported causative relationship between perineal talcum powder use and ovarian cancer.

4. The theories as to how talc or asbestos would reach the ovaries have not been validated, and the scientific community has repeatedly expressed the opinion that the potential mechanism by which talcum powder is associated with ovarian cancer remains speculative.

5. Additional Bradford Hill factors – temporality, coherence of the association and analogy – are not satisfied based on the available epidemiologic evidence and do not support the allegation that talcum powder use can cause ovarian cancer.

6. To the extent plaintiffs' experts opine that asbestos is an accessory mineral present in cosmetic talc that causes ovarian cancer, this theory would not alter the analysis because the existing epidemiological literature regarding perineal talc use would necessarily account for the presence of any asbestos in the products used in those studies. Plaintiffs' experts' asbestos-based theories are also problematic due to the lack of a plausible mechanism by which asbestos could reach the ovaries and a lack of any reliable epidemiology supporting such a causal connection.

IV. APPROACH

A. Bradford Hill Framework

Epidemiologists and other scientists are often tasked with determining whether or not an exposure can cause an illness or condition. After an association has been demonstrated, criteria articulated by Austin Bradford Hill in a lecture in 1965 are often employed. These Bradford Hill considerations, or criteria, are considered the gold standard for assessing causation based on observed associations. The nine considerations are: consistency, strength of association, specificity, dose-response relationship, temporality, biologic plausibility, coherence of the association, analogy and experimentation.[1] In applying these criteria, an epidemiologist should consider all available evidence, which can be assessed and graded according to its sufficiency (or lack thereof) to establish a causal link. Evidence typically comes from research studies that involve humans, but it can also include well-designed studies of animals or in vitro systems (toxicological and experimental) to provide supportive evidence, especially for plausibility.

Another useful factor for assessing causation includes consideration of non-causal explanations for the results of individual studies.[2] As explained further below, these other explanations can come from bias, confounding and chance. For example, drinking coffee might be correlated with a higher risk of lung cancer, but the cause of the additional cases of lung cancer among individuals who drink coffee would be smoking cigarettes. In this example, the obvious confounding factor is that individuals who drink coffee are more likely to smoke. But confounding factors are not always identifiable, even after extended study, and these and other factors can consistently drive statistical associations that are not causal in nature. Such limitations can be quite important, as they can lead to risk estimates that are falsely higher or lower than actual risk, and they can even lead to conclusions that an exposure causes a disease when it does not, and vice versa.

B. Methodology

I was asked to assess whether perineal exposure to talcum powder causes ovarian cancer. Based on my extensive qualifications and experience, review of the available studies and data and assessment of the Bradford Hill factors, I conclude that the observations and evidence to date are insufficient to find a causal relationship between perineal exposure to talcum powder and ovarian cancer.

My opinions are based on a review of the epidemiology literature relevant to the evaluation of the association between perineal talcum powder use and ovarian cancer. In my review, I considered case-control studies, prospective cohort studies and meta-analyses. I did not consider randomized trial data, since I am not aware of any such data reporting on the presence

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[1] Hill AB. The Environment and Disease: Association or Causation? Proc R Soc Med. 1965; 58(5):295-300 ("Hill 1965").

[2] Elwood JM. Causal Relationships in Medicine: A Practical System for Critical Appraisal. Oxford: 1988, 163-182.

or absence of an association between of talcum powder and ovarian cancer. Because the accuracy of the findings of case-control and cohort studies can be influenced by bias and confounders, I carefully considered whether there was any indication that these sorts of errors affected the results.

In evaluating the epidemiologic data and other scientific evidence under the Bradford Hill framework, I primarily focus on whether the criteria of strength of association, consistency of the association, biologic gradient (or dose response) and biologic plausibility have been met. Although it is not essential to address every factor under the Bradford Hill framework, as plaintiffs' experts acknowledge,[3] I also address specificity, temporality, coherence of the association, experiment and analogy.

Lastly, I reviewed several of the reports submitted by plaintiffs' experts and their depositions. A number of these experts claim to have analyzed the Bradford Hill criteria and to have concluded through these analyses that perineal talc use causes ovarian cancer. I assess and address several of plaintiffs' experts' methods and analyses in this regard.

V. STUDY DESIGNS

Epidemiologists recognize that there is a hierarchy of evidence with respect to human studies. Clinical trials are often considered the strongest type of evidence, followed by observational studies (cohort and case-control). The lowest quality of evidence comes from case reports, case series and descriptive studies.[4]

There are two main types of epidemiological studies at issue here: prospective cohort studies and case-control studies.

Prospective cohort studies consist of identifying a large group of healthy individuals who differ in the key areas being observed and following them forward in time. Based on the data collected, it is determined how the factors of interest, e.g., exposure to talcum powder, are associated with a certain outcome or disease. Cohort studies are widely regarded as more reliable than retrospective case-control studies because they are not susceptible to recall bias, which is the propensity of study subjects with the disease that is being studied to inaccurately report their exposure to the agent at issue, a phenomenon that can generate inflated risk estimates.[5] Cohort studies generally avoid this pitfall because they are prospective rather than retrospective.[6] Due to the ability of cohort studies to assess exposure at baseline instead of relying solely on recall, they can be better suited to detect risks from exposure to an agent.

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[3] Smith-Bindman Rep. at 36; Singh Rep. at 62.

[4] Elwood at 174-175.

[5] Gertig DM, Hunter DJ, Cramer DW, et al. Prospective Study of Talc Use and Ovarian Cancer. J Natl Cancer Inst. 2000; 92(3): 249-252, 252 ("Gertig 2000"); Langseth H, Hankinson SE, Siemiatycki J, Weiderpass E. Perineal Use of Talc and Risk of Ovarian Cancer. J Epidemiol Community Health. 2008; 62(4):358-360, 358 ("Langseth 2008"). *See generally* Leon Gordis. Epidemiology. 5th ed. Philadelphia, PA: 2014.

[6] Although there are also retrospective cohort studies, those are not at issue here, because the cohort studies involving cosmetic talc use are prospective in design.

In case-control studies, individuals with the disease of interest (cases) and those without the disease of interest (controls) are first identified. These two groups are then compared to assess any differences between them regarding a specified exposure. Case-control studies can be further broken down into population-based and hospital-based studies. Hospital-based studies draw their control population from patients who are hospitalized with conditions other than the one under study. Population-based studies draw study participants from the general population.

VI. REVIEW OF EPIDEMIOLOGY DATA

In forming my opinions, I employed search tools, including Medline and Google Scholar, to identify studies that examined the association of perineal talcum powder use and ovarian cancer. I also reviewed the reference lists of individual studies and the meta-analyses to assemble a complete list of studies. Specifically, I first located and reviewed the relevant cohort studies, meta-analyses and case-control epidemiologic studies. I then reviewed how other medical experts or other professional organizations interpreted those studies. My reliance list, which is attached to this report, is comprised of all studies located and assessed specifically for this case. In total, I identified and reviewed 32 case-control studies and three prospective cohort studies published since 1982 that pertain to perineal talc use and ovarian cancer.

It is my understanding that plaintiffs are asserting in this litigation that talc products contain asbestos. The epidemiological literature concerning talc products and ovarian cancer generally has not attempted to investigate the question whether asbestos is present in talc as an accessory mineral. Nevertheless, if talc products have generally contained asbestos, the epidemiological literature would reflect the risks of asbestos in talc.

A. Strength Of Association Is Weak.

The first Bradford Hill criterion, strength of the association, refers to the magnitude of the risk of developing a given outcome in the presence of a measured risk factor. In the studies discussed in this report, risk is reported in various ways – as a relative risk ("RR"), odds ratio ("OR"), or hazard ratio ("HR") – typically with a confidence internal ("CI"). A relative risk "of an event is the likelihood of its occurrence after exposure to a risk variable" – here, talcum powder or asbestos – "as compared with the likelihood of its occurrence in a control or reference group."[7] An odds ratio is "a comparison of the odds of an event after exposure to a risk factor with the odds of that event in a control or reference situation."[8] A hazard ratio is a type of relative risk that measures "how often a particular event happens in one group compared to how often it happens in another group, over time."[9] In each case, the risk is expressed as a number for which 1 is the denominator, so that a relative risk of 1.3, for example, would mean that the outcome of interest occurred 1.3 times as often in the exposed group as compared to the control

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[7] Andrade C. Understanding Relative Risk, Odds Ratio, and Related Terms. J Clin Psychiatry. 2015; 76(7):e857-861.

[8] *Id.*

[9] National Cancer Inst., NCI Dictionary of Cancer Terms, "hazard ratio," https://www.cancer.gov/publications/dictionaries/cancer-terms/def/hazard-ratio.

group – a 30% greater incidence. A relative risk of 1.0, by contrast, would mean there was no difference. In each case, a confidence interval can be calculated to determine statistical significance – in essence, whether the difference between the exposed and unexposed groups is likely to persist if the same study were repeated. When a confidence interval contains 1.0, the result is deemed not to be statistically significant because the possibility that there is no real association is within the expected range of results. It is typical to calculate a 95% confidence interval, expressed in this report as "95% CI," meaning that if the study were repeated, the results would be expected to fall within the confidence interval 95% of the time.

While there is no absolute cutoff to define a large versus a small relative risk, Hill provided examples of large risks, including the 200 times risk of scrotal cancer in chimney sweeps, an estimate of 9-10 times risk of lung cancer in smokers and 20-30 times risk of lung cancer in heavy smokers. As an example of a low risk, Dr. Hill used death from coronary thrombosis in smokers, which he described as "no more than twice, probably less" than the death rate in non-smokers. Dr. Hill further explained:

> "[T]hough there is good evidence to support causation it is surely much easier in this case to think of some features of life that may go hand in hand with smoking—features that might conceivably be the real underlying cause or, at the least, an important contributor, whether it be lack of exercise, nature of diet or other factors. But to explain the pronounced excess in cancer of the lung in any other environmental terms requires some feature of life so intimately linked with cigarette smoking and with the amount of smoking that such a feature should be easily detectable."[10]

What this passage from Hill means is that low observed risks are more likely to be non-causal than are high risks, because the effects of distorting factors (such as confounders and bias) have a greater chance of being the true explanation for the observations. Because very small risks are obviously highly susceptible to distorting effects in observational studies, further evidence is required to demonstrate that the purported association did not arise from bias, confounding or chance alone. Plaintiffs' experts express opinions about risks articulated as approximately a 1.2-1.3 odds ratio.[11] This is considered a weak association by the scientific community, as some of plaintiffs' experts acknowledge.[12] To the extent other plaintiffs' experts dispute this point (most notably Dr. Moorman, who attempted to argue that straightforward adjectives like "weak," "modest" or "strong" do not have "clear definitions"), their position is simply not credible, and even Dr. Moorman acknowledges that 1.2-1.3 is "weaker" than well-established large associations such as smoking/lung cancer.[13] While the size of the risk does not, in itself, determine causation, this purported low risk estimate is not strong evidence of

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[10] Hill 1965.

[11] Moorman Rep. at 17.

[12] Singh Dep. 140:19-25 (agreeing that scientific literature does not consider 1.3 a strong association).

[13] Moorman Dep. 246:24-250:16; *id.* 287:14-289:3 (refusing to define a "weak association" but acknowledging that epidemiology textbooks would not agree that it cannot be defined); *see also id.* 145:4-17 (agreeing that the medical community accepts smoking as a cause of lung cancer but questioning the definition of "medical community" when asked the same about talc and ovarian cancer).

causation. As plaintiffs' expert, Dr. Siemiatycki, wrote in a 1988 article, "[s]mall excess relative risks, even if they are statistically significant, are often interpreted with great caution, if not skepticism."[14]

1. Results Of Cohort Studies, Case-Control Studies And Meta-Analyses And Pooled Studies

As fully set forth in the next sections, the prospective epidemiological studies (cohort studies) do not show a statistically significant association between genital talc use and ovarian cancer, while a subset of the population-based case-control studies do show weak statistically significant associations.

a. Results of Cohort Studies

The most recent cohort study, referred to by many as the "Sister Study," enrolled 50,884 women in the U.S. and Puerto Rico beginning in 2003, who had a sister diagnosed with breast cancer, and followed 41,654 of those women for a median 6.5 years.[15] The study identified 154 cases of ovarian cancer and found no association between the use of talc and ovarian cancer – in fact, there was an inverse association that was not statistically significant (HR 0.73 (95% CI: 0.44-1.2)).[16] Of note, this study separately found an association between douching and ovarian cancer, suggesting that douching (which sometimes accompanies perineal talc use) may be a confounding variable that has not sufficiently been accounted for in past studies.[17]

A prior cohort study known as the Women's Health Initiative Study followed 61,576 women for a mean of 12.4 years.[18] The study showed no increased risk of ovarian cancer from genital use of talc (HR 1.12 (95% CI: 0.92-1.36)), no increased risk of ovarian cancer from genital talc use for 10 or more years (HR 0.98 (95% CI: 0.75-1.29)) or 20 or more years (HR 1.10 (95% CI: 0.82-1.48)), and no increased risk of ovarian cancer with talc use on sanitary napkins (HR 0.95 (95% CI: 0.76-1.20)) or contraceptive diaphragms (HR 0.92 (95% CI: 0.68-1.23)).[19] The result for combined powder use was a statistically non-significant hazard ratio (HR 1.06 (95% CI: 0.87-1.28)) and an even lower statistically non-significant hazard ratio for combined use for more than ten years (HR 1.02 (95% CI: 0.80-1.30)).[20] The authors concluded

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[14] Siemiatycki Dep. 328:22-329:2.

[15] Gonzalez NL, O'Brien KM, D'Aloisio AA, Sandler DP, Weinberg CR. Douching, Talc Use, and Risk of Ovarian Cancer. Epidemiology. 2016; 27(6): 797–802. ("Gonzalez 2016").

[16] *Id.* at 800-02.

[17] *Id.* at 800.

[18] Houghton SC, Reeves KW, Hankinson SE, et al. Perineal Powder Use and Risk of Ovarian Cancer. J Natl Cancer Inst. 2014; 106(9): dju208. ("Houghton 2014").

[19] *Id.*

[20] *Id.*

that "perineal powder use does not appear to influence ovarian cancer risk."[21]

The results of an additional cohort study were published in 2000[22] and updated in another publication ten years later.[23] These reports looked at talc use within the Nurses' Health Study ("NHS"), which was a prospective cohort of 121,700 registered nurses in the United States and was established in 1976.[24] The Gertig analysis showed no statistically significant association between perineal talc use (RR 1.09 (95% CI: 0.86-1.37)), use of talc on sanitary napkins (RR 0.89 (95% CI: 0.61-1.28)) and for both uses combined (RR 0.90 (95% CI: 0.59-1.37)).[25] It further showed no statistically significant association for various different frequencies of use and no indication that risk increased with more frequent use: less than one per week (RR 1.14 (95% CI: 0.81-1.59)); 1-6 uses per week (RR 0.99 (95% CI: 0.67-1.46)); daily use (RR 1.12 (95% CI: 0.82-1.55)).[26] When examining the results by histology, the authors observed a weak statistically significant association for serous invasive (RR 1.40 (95% CI: 1.02-1.91)) but no other types of ovarian cancer.[27] They noted that perineal talc use "may modestly increase the risk of invasive serous ovarian cancers" but not for "all serous cancers (including borderline cancers), endometrioid cancers, or mucinous cancers," and concluded overall that their "results provide little support for any substantial association between perineal talc use and ovarian cancer risk."[28]

The 2010 Gates report, which followed up on the Nurses' Health cohort ten years later, found no statistically significant elevations in risk for talc use for all epithelial ovarian cancers (RR 1.06 (95% CI: 0.89-1.28)), serous invasive ovarian cancers (RR 1.06 (95% CI: 0.84-1.35)), endometrioid ovarian cancers (RR 1.06 (95% CI: 0.66-1.69)), or mucinous ovarian cancers (RR 1.50 (95% CI: 0.84-2.66)).[29] The authors concluded that their results for talc exposure "generally are consistent with the existing literature," i.e., consistent with generally null and/or weakly associated results.[30] It is notable too, that with further passage of time, there was no longer an increased association for the serous invasive type of ovarian cancer.

Plaintiffs' experts' argument that the Gates report should be disregarded because the participants in the Nurses' Health Study were only asked about talcum powder use once is

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21 *Id.*

22 Gertig 2000.

23 Gates MA, Rosner BA, Hecht JL, Tworoger SS. Risk Factors for Epithelial Ovarian Cancer by Histologic Subtype. Am J Epidemiol. 2010; 171(1):45-53, 50 ("Gates 2010").

24 Gertig 2000.

25 *Id.* at 251.

26 *Id.*

27 *Id.*

28 *Id.* at 250-51.

29 Gates 2010 at 50.

30 *Id.* at 51. While the 2010 NHS updated the number of women studied, the new participants were not asked about talc, which the authors acknowledged was a "weakness[]" in the study. *Id.* at 52.

unfounded.[31] Ten additional years of follow-up is valuable data regardless of whether further questioning regarding talc use took place. Moreover, as other studies and plaintiffs' experts themselves have admitted, for women who are ever-users of perineal talcum powder, the mean duration of use is greater than 20 years[32] and the vast majority of women who use talcum powder initiate use before age 36.[33] That means that, even though the participants were only asked about their talcum powder use once, the data collected on perineal talcum powder application would have likely reflected chronic, habitual use. For similar reasons, recent meta-analyses by Penninkilampi (relied on heavily by plaintiffs' experts)[34] and Taher[35] (discussed further below) are of questionable value in light of their omission of the findings reported by Gates, which are derived from a cohort study that found no statistically significant elevations in risk for talc users with respect to epithelial ovarian cancers, serous invasive ovarian cancers, endometrioid ovarian cancers or mucinous ovarian cancers.

Dr. McTiernan's argument that cohort studies are limited because they were "designed to study a large number of outcomes and a wide variety of exposures" in addition to talc and ovarian cancer[36] is also wrong. The fact that cohort studies are able to study many variables and outcomes is an illustration of what is valuable and can be achieved with cohort studies. I know of no epidemiologists who believe that the results of all cohort studies should be discounted due to this common design trait; indeed, such a view would conflict with the generally accepted principle that cohort studies can produce a higher level of evidence than case-control studies. Moreover, the typical concern when studies include multiple variables is that they might report false positive associations for particular variables, and no plaintiffs' expert argues that the talc results in cohort studies are false positives (although that argument could be applied to the single positive finding from the Gertig study). Dr. McTiernan relatedly argues that the cohort studies were not "able to accurately measure dose of exposure," but this is equally true of case-control studies, as discussed herein.[37]

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[31] Singh Dep. 164:16-23; Moorman Dep. 190:4-24; McTiernan Dep. 224:3-7; Smith-Bindman Rep. at 20.

[32] Wu AH, Pearce CL, Tseng CC, Pike MC. African Americans and Hispanics Remain at Lower Risk of Ovarian Cancer Than Non-Hispanic Whites after Considering Nongenetic Risk Factors and Oophorectomy Rates. Cancer Epidemiol Biomarkers Prev. 2015; 24(7):1094-100 ("Wu 2015").

[33] Singh Dep. 165:2-8; Gates MA, Tworoger SS, Terry KL, et al. Talc use, variants of the GSTM1, GSTT1, and NAT2 genes, and risk of epithelial ovarian cancer. Cancer Epidemiol Biomarkers Prev. 2008; 17(9):2436-2444 ("Gates 2008").

[34] Penninkilampi R, Eslick GD. Perineal Talc Use and Ovarian Cancer: A Systematic Review and Meta-Analysis. Epidemiology. 2018; 29(1):41-49, 44 ("Penninkilampi 2018"); Smith-Bindman Rep. at 27 ("Penninkilampi provides a comprehensive and high quality review"); McTiernan Rep. at 49 ("[T]he results of this 2018 meta-analysis give strong support for an association between perineal talcum powder product use and risk for ovarian cancer.").

[35] Taher MK, Farhat N, Karyakina NA, et al. Systematic Review and Meta-Analysis of the Association Between Perineal Use of Talc and Risk of Ovarian Cancer (2018) (unpublished manuscript) ("Taher 2018").

[36] McTiernan Rep. at 46.

[37] *Id.*

Plaintiffs' experts also criticize cohort studies for having short follow-up and therefore supposedly not considering the latency period for ovarian cancer.[38] In light of the data noted above about mean initiation and duration of talc use, it is reasonable to assume that the date on which study participants were asked about their talcum powder use was not the date of first use and thus not the date that a true latency period would have begun. Moreover, the Women's Health Initiative Study asked about talcum powder use for 20-plus years and found no statistically significant increased risk in ovarian cancer after following those women for 12.4 years (meaning at least 32.4 years of latency were factored in),[39] and the Sister Study enrolled women between the ages of 35-74 and followed up after 6 years.[40] Therefore, it is clear in the case of the WHI study, and quite likely in the case of the Sister Study, that substantial numbers of cohort study participants were using talcum powder for decades, long enough to put any serious concerns about latency to rest.

Any criticism of the studies that rests on the idea of a latency period is highly speculative anyway. For the reasons set out in this report, science has not even established a causal relationship between talc and ovarian cancer of any sort; far less has it established a latency effect or the duration of any such effect. There is simply no scientific basis for the suggestion of a number of plaintiffs' experts that it takes 20 years for some unspecified degree of perineal talc exposure to cause ovarian cancer.

Finally, plaintiffs' experts' criticisms of cohort studies are collectively suspect because they are so extensive when compared to their relatively muted criticisms for case-control studies, which, as I detail in the next sections, have their own significant weaknesses. For example, Dr. Smith-Bindman devotes several pages of her report to lodging numerous criticisms of each study that reported on cohort data; although she mostly spares Gertig 2000 (which happens to be the one cohort study she believes supports her theory), she declares in summary fashion that there is nothing "meaningful" to be gleaned from any of the other cohort studies.[41] Yet she provides no similar analysis of the strengths and weaknesses of the case-control studies, noting in the single paragraph in which she discusses them that her review and abstraction of data from them was done "[w]ithout assessing the[ir] quality."[42] Similarly, Dr. Moorman did not offer any criticisms or cautions regarding the talc meta-analyses, whereas she pointed out limitations of cohort studies extensively in her report.[43] None of the studies is perfect. But plaintiffs' experts' focused attack on cohort studies (as they seek to minimize the significant flaws of the case-control studies) reveals the biased and unscientific nature of their analyses.

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38 Singh Rep. at 11, 53; McTiernan Rep. at 47.

39 Houghton 2014 at 2.

40 Gonzalez 2016 at 2.

41 Smith-Bindman Rep. at 20-22.

42 *Id.* at 29-30.

43 Moorman Dep. 164:16-18; Moorman Rep. at 24-28.

In summary, none of the cohort studies found a statistically significant association between talc use and ovarian cancer.[44] The fact that these studies have shown uniformly null results indicates no association between talc use and ovarian cancer.

b. Results of Case-Control Studies

I have identified 25 population-based case-control studies addressing talc use and ovarian cancer.[45] The following table sets forth these studies' findings with respect to the association between ever/never talc use and ovarian cancer:

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[44] Berge W, Mundt K, Luu H, Boffetta P. Genital Use of Talc and Risk of Ovarian Cancer: A Meta-Analysis. Eur J Cancer Prev. 2018; 27(3):248-257, 251 ("Berge 2018") (assigning a statistically insignificant 1.02 relative risk to the cohort studies in aggregate).

[45] Cramer DW, Welch WR, Scully RE, Wojciechowski CA. Ovarian cancer and talc: a case-control study. Cancer. 1982; 50(2):372-376; Harlow BL, Weiss NS. A case-control study of borderline ovarian tumors: the influence of perineal exposure to talc. Am J Epidemiol. 1989; 130(2):390-394; Harlow BL, Cramer DW, Bell DA, Welch WR. Perineal exposure to talc and ovarian cancer risk. Obstet Gynecol. 1992; 80(1):19-26 ("Harlow 1992"); Chen Y, Wu PC, Lang JH, et al. Risk factors for epithelial ovarian cancer in Beijing, China. Int J Epidemiol. 1992; 21(1):23-29; Cramer DW, Xu H. Epidemiologic evidence for uterine growth factors in the pathogenesis of ovarian cancer. Ann Epidemiol. 1995; 5(4):310-314; Purdie D, Green A, Bain C, et al. Reproductive and other factors and risk of epithelial ovarian cancer: an Australian case-control study. Survey of Women's Health Study Group. Int J Cancer. 1995; 62(6):678-684; Chang S, Risch HA. Perineal Talc Exposure and Risk of Ovarian Carcinoma. Cancer. 1997; 79(12):2396-2401 ("Chang & Risch 1997"); Cook LS, Kamb ML, Weiss NS. Perineal Powder Exposure and the Risk of Ovarian Cancer Am J Epidemiol. 1997; 145(5):459-465 ("Cook 1997"); Green A, Purdie D, Bain C, et al. Tubal sterilisation, hysterectomy and decreased risk of ovarian cancer. Survey of Women's Health Study Group. Int J Cancer. 1997; 71(6):948-951; Godard B, Foulkes WD, Provencher D, et al. Risk factors for familial and sporadic ovarian cancer among French Canadians: a case-control study. Am J Obstet Gynecol. 1998; 179(2):403-410; Cramer DW, Liberman RF, Titus-Ernstoff L, et al. Genital Talc Exposure and Risk of Ovarian Cancer. Int J Cancer. 1999; 81(3):351-356 ("Cramer 1999"); Ness RB, Grisso JA, Cottreau C, et al. Factors related to inflammation of the ovarian epithelium and risk of ovarian cancer. Epidemiology. 2000; 11(2):111-117; Mills PK, Riordan DG, Cress RD, Young HA. Perineal talc exposure and epithelial ovarian cancer risk in the Central Valley of California. Int J Cancer. 2004; 112(3):458-464 ("Mills 2004"); Cramer DW, Titus-Ernstoff L, McKolanis JR, et al. Conditions associated with antibodies against the tumor-associated antigen MUC1 and their relationship to risk for ovarian cancer. Cancer Epidemiol Biomarkers Prev. 2005; 14(5):1125-1131; Jordan SJ, Green AC, Whiteman DC, Webb PM, Australian Ovarian Cancer Study Group. Risk factors for benign, borderline and invasive mucinous ovarian tumors: epidemiological evidence of a neoplastic continuum? Gynecol Oncol. 2007; 107(2):223-230; Gates 2008; Merritt MA, Green AC, Nagle CM, et al. Talcum powder, chronic pelvic inflammation and NSAIDs in relation to risk of epithelial ovarian cancer. Int J Cancer. 2008; 122(1):170-176 (2008) ("Merritt 2008"); Moorman PG, Palmieri RT, Akushevich L, et al. Ovarian cancer risk factors in African-American and white women. Am J Epidemiol. 2009; 170(5):598-606; Wu AH, Pearce CL, Tseng CC, et al. Markers of inflammation and risk of ovarian cancer in Los Angeles County. Int J Cancer. 2009; 124(6):1409-1415 ("Wu 2009"); Rosenblatt KA, Weiss NS, Cushing-Haugen KL. Genital powder exposure and the risk of epithelial ovarian cancer. Cancer Causes Control. 2011; 22(5):737-742 ("Rosenblatt 2011"); Kurta ML, Moysich KB, Weissfeld JL, et al. Use of fertility drugs and risk of ovarian cancer: results from a U.S.-based case-control study. Cancer Epidemiol Biomarkers Prev. 2012; 21(8):1282-1292; Kotsopoulos J, Terry KL, Poole EM, et al. Ovarian cancer risk factors by tumor dominance, a surrogate for cell of origin. Int J Cancer. 2013; 133(3):730-739; Wu 2015; Cramer DW, Vitonis AF, Terry KL, et al. The Association Between Talc Use and Ovarian Cancer: A Retrospective Case-Control Study in Two US States. Epidemiology. 2016; 27(3):334-346 ("Cramer 2016"); Schildkraut JM, Abbott SE, Alberg AJ, et al. Association between Body Powder Use and Ovarian Cancer: The African American Cancer Epidemiology Study (AACES). Cancer Epidemiol Biomarkers Prev. 2016; 25(10):1411-1417 (2016) ("Schildkraut 2016").

Author, Year	Ever/Never Results
Cramer 1982	RR 1.92 (95% CI: 1.27-2.89)
Harlow & Weiss 1989	RR 1.10 (95% CI: 0.70-2.10)
Harlow 1992	OR 1.50 (95% CI: 1.00-2.10)
Chen 1992	RR 3.90 (95% CI: 0.90-10.6)
Cramer & Xu 1995	OR 1.10 (95% CI: 0.60-2.10)
Purdie 1995	OR 1.27 (95% CI: 1.04-1.54)
Chang & Risch 1997	OR 1.42 (95% CI: 1.08-1.86)
Cook 1997	RR 1.60 (95% CI: 0.90-2.80)
Green 1997	RR 1.30 (95% CI: 1.10-1.60)
Godard 1998	RR 2.49 (95% CI: 0.94-6.58)
Cramer 1999	OR 1.45 (95% CI: 0.97-2.18)
Ness 2000	OR 1.50 (95% CI: 1.10-2.00)
Mills 2004	OR 1.37 (95% CI: 1.02-1.85)
Cramer 2005	OR 1.16 (95% CI: 0.90-1.49)
Jordan 2007	OR 1.00 (95% CI: 0.40-2.10)
Gates 2008	RR 1.36 (95% CI: 1.14-1.63)
Merritt 2008	OR 1.17 (95% CI: 1.01-1.36)
Moorman 2009	Afr. Am.: OR 1.19 (95% CI: 0.68-2.09) Caucasian: OR 1.04 (95% CI: 0.82-1.33)
Wu 2009	RR 1.53 (95% CI: 1.13-2.09)
Rosenblatt 2011	OR 1.27 (95% CI: 0.97-1.66)
Kurta 2012	OR 1.40 (95% CI: 1.16-1.69)
Kotsopoulos 2013[46]	RR 1.19 (95% CI: 0.73-1.96)
Wu 2015	OR 1.46 (95% CI: 1.27-1.69)
Cramer 2016	OR 1.33 (95% CI: 1.16-1.52)
Schildkraut 2016	OR 1.44 (95% CI: 1.11-1.86)

I have identified seven hospital-based case-control studies addressing the association between talc use and ovarian cancer.[47] As set forth in the following table, none of these studies observed a statistically significant association:

[46] Study looked at all types of genital powder used at least once per week.

[47] Hartge P, Hoover R, Lesher LP, McGowan L. Talc and Ovarian Cancer. JAMA. 1983; 250(14):1844 ("Hartge 1983"); Whittemore AS, Wu ML, Paffenbarger RS Jr, et al. Personal and environmental characteristics related to epithelial ovarian cancer. II. Exposures to talcum powder, tobacco, alcohol, and coffee. Am J Epidemiol. 1988; 128(6):1228-1240 ("Whittemore 1988"); Booth M, Beral V, Smith P. Risk factors for ovarian cancer: a case-control study. Br J Cancer. 1989; 60(4):592-598 ("Booth 1989"); Rosenblatt KA, et al. Mineral fiber exposure and the development of ovarian cancer. Gynecol Oncol. 1992; 45(1):20-25. ("Rosenblatt 1992"); Tzonou A, Polychronopoulou A, Hsieh CC, et al. Hair dyes, analgesics, tranquilizers and perineal talc application as risk factors for ovarian cancer. Int J Cancer. 1993; 55(3):408-410 ("Tzonou 1993"); Hartge P, Stewart P. Occupation and ovarian cancer: a case-control study in the Washington, DC, metropolitan area, 1978-1981. J Occup Med. 1994; 36(8):924-927 ("Hartge & Stewart 1994"); Wong C, Hempling RE, Piver MS, et al. Perineal talc exposure and subsequent epithelial ovarian cancer: a case-control study. Obstet Gynecol. 1999; 93(3):372-376. ("Wong 1999").

Author, Year	Ever/Never Results
Hartge 1983	RR 0.70 (95% CI: 0.40-1.10)
Whittemore 1988	RR 1.45 (95% CI: 0.81-2.60)
Booth 1989	RR 1.30 (95% CI: 0.80-1.90)
Rosenblatt 1992	OR 1.70 (95% CI: 0.70-3.90)
Tzonou 1993	RR 1.05 (95% CI: 0.28-3.98)
Hartge & Stewart 1994	RR 0.3 (95% CI: 0.1-1.4) to RR 0.5 (95% CI: 0.2-1.5)[48]
Wong 1999	OR 1.00 (95% CI: 0.80-1.30)

In summary, 11 of the 25 population-based case-control studies do not show a statistically significant association, and none of the hospital-based studies does. Notably, the authors of the case-control studies have generally cautioned that even when they found a statistically significant elevated risk, their results do not establish causation, even in combination with the results of other studies.[49]

c. Results of Meta-analyses and Pooled Studies

Meta-analyses and pooled studies, which use statistical methods to pool results from different studies, have also been performed on the body of talc-ovarian cancer epidemiological literature. These studies have calculated an overall odds ratio of approximately 1.3,[50] which they have characterized as a "relatively weak odds ratio[]" that "can be attributed to bias in" case-control studies.[51] As some of these studies have stated, the epidemiological data are "insufficient

[48] This study did not provide a value for ever/never use; range reflects values across three strata of use durations.

[49] Cramer DW, Welch WR, Berkowitz RS, Godleski JJ, Presence of talc in pelvic lymph nodes of a woman with ovarian cancer and long-term genital exposure to cosmetic talc. Obstet Gynecol. 2007; 110(2 Pt 2):498-501, 500 (case study stating that "[w]e are not claiming that a causal relationship between ovarian cancer and talc use is proven for this case or in general").

[50] Berge 2018 at 251 (RR 1.22 (95% CI: 1.13-1.30)); Terry KL, Karageorgi S, Shvetsov YB, et al. Genital Powder Use and Risk of Ovarian Cancer: A Pooled Analysis of 8,525 Cases and 9,859 Controls. Cancer Prev Res (Phila). 2013; 6(8):811-821 ("Terry 2013") (OR 1.24 (95% CI: 1.15-1.33)); Langseth 2008 (OR 1.40 (95% CI: 1.29-1.52)).

[51] Berge 2018 at 253; Cramer DW, Liberman RF, Titus-Ernstoff L, et al. Genital Talc Exposure and Risk of Ovarian Cancer. Int J Cancer. 1999; 81(3):351-356, 354 ("Cramer 1999"); Huncharek M, Geschwind JF, Kupelnick B. Perineal application of cosmetic talc and risk of invasive epithelial ovarian cancer: a meta-analysis of 11,933 subjects from sixteen observational studies. Anticancer Res. 2003; 23(2C):1955-1960 (2003 meta-analysis explaining that "[s]election bias and uncontrolled confounding may account for the positive associations seen in prior epidemiological studies"); Rothman KJ, Pastides H, Samet J. Interpretation of Epidemiologic Studies on Talc and Ovarian Cancer 4 (Nov. 28, 2000), https://ntp.niehs.nih.gov/ntp/newhomeroc/roc12/mcewen-07-14-04.pdf ("Recall bias can readily introduce enough bias to produce the modestly-sized overall effect (RR = 1.3) that emerges from these studies."). I am aware of Dr. Zambelli-Weiner's criticisms of the Huncharek studies, but Dr. Zambelli-Weiner does not claim that the studies understated the association between genital talc use and ovarian cancer; indeed, her efforts to replicate the dose-response calculations in Huncharek (2003) similarly failed to show a dose-response relationship.

to establish a causal association between perineal use of talc and ovarian cancer risk" and "not support[ive of] a causal interpretation of the association."[52]

Plaintiffs' experts rely on a 2018 study by Penninkilampi and Eslick, which conducted a literature review of studies addressing talcum powder use and ovarian cancer and performed a meta-analysis that "revealed an increased risk of ovarian cancer associated with any perineal use of talc (. . . OR = 1.31; 95% CI = 1.24, 1.39)."[53] Although the finding was statistically significant, it remains low, with a 1.31 odds ratio that falls within the range of prior studies, adding little to the existing literature on this question. Indeed, the authors acknowledged that several meta-analyses had been conducted by 2018, but sought to justify the need for another in light of ongoing litigation, contending that "the association between talc use and ovarian cancer [has taken] on considerable relevance" because "Johnson & Johnson has recently had damages levied to the total of US$717 million against [it] in five law suits" and because "producers of talcum powder products continue to sell these products without any warning labels regarding perineal use and potential associations with ovarian cancer," leading the authors to conclude that "there is a need for clarification, to allow women to be adequately informed of the risk of use of these products, possibly preventing future harm."[54] This is an unusual statement in a scientific article and especially odd in an article that is ostensibly premised on the idea that existing science has not concretely defined the risk that the authors are suggesting should be warned against. The study is also puzzling in that its stated purpose is to update prior meta-analyses – in particular, because "the results of a number of large case-control studies and two cohort studies" had been reported since the last meta-analysis was published[55] – and yet the meta-analyses wholly excluded consideration of the Gates report (the NHS follow-up), another cohort study published during the same period. Ultimately, notwithstanding the authors' expressed concerns about warning women and updating the research, their conclusions echo those of prior studies, acknowledging in some detail the possibility that recall bias drove the results in the case-control studies[56] and concluding that while the authors believe their results are "suggestive of a causal association," it remains the case that "[a]dditional epidemiologic evidence from prospective studies with attention to effects within ovarian cancer subtype is warranted" and that "it is important that research into this association continue."[57]

I also note that plaintiffs' experts Dr. Smith-Bindman and Dr. Siemiatycki decided to conduct their own meta-analyses for purposes of their reports. I did not attempt such an undertaking because there is no need; there have been a number of recent meta-analyses in this area, and not enough new recent studies to justify running the same meta-analysis one more time

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52 Langseth 2008 at 359; Berge 2018 at 256.

53 Penninkilampi 2018 at 44.

54 *Id.* at 42.

55 *Id.*

56 *Id.* at 47.

57 *Id.* at 48.

(indeed, Dr. Siemiatycki acknowledges that the cumulative 1.28 relative risk his analysis generated is on par with those of the recent published meta-analyses).[58]

Dr. Smith-Bindman purports to have conducted her meta-analysis in an effort to specifically assess whether "regular" talc use causes ovarian cancer and serous invasive ovarian cancer in particular.[59] According to Dr. Smith-Bindman, a "narrow[er]" meta-analysis would offer the "most meaningful and consistent results," ostensibly by reducing variation between the included studies as to "relevant factors such as age or race/ethnicity."[60] But she does not cite any reference in support of her "less is more" theory; nor does she identify any generally accepted criticisms of existing talc meta-analytic work that would justify her narrower approach. She also offers no basis for concluding that the results of her study are somehow more reliable than the studies that have previously been done on the same body of literature, and which have been published after peer review. And indeed they are not, due to at least the following significant methodological deficiencies:

- Dr. Smith-Bindman states that she chose to focus on serous invasive ovarian cancer because it was the only subtype "for which most individual research studies accumulated sufficient cases for valid statistical analysis," but she provides no analysis or data to support this claim.[61] Her decision to focus on serous invasive ovarian cancer (the only subtype that previously has been associated with an increased risk from talc in any of the cohort studies) illustrates a systematic exclusion of data that do not support her theory.

- Her concession that her measure of "regular" talc use was "subjective[]" is an understatement.[62] She defines "regular use" "ideally as daily or at least more than 3 uses per week," but she also "accepted studies that defined use as 'regular' where the description made it clear that this was regular use."[63] For some studies that reported "regular" use but sub-grouped that categorization, she only "included data for women in the highest use category," and only if that group "was large enough to be meaningful."[64] And when studies "asked about ever use but defined use and stratified results by use," she "included any data that may have reflected daily use."[65] Far too many questions arise from these vague and subjective criteria. For example, why did Dr. Smith-Bindman arbitrarily choose three uses per week as the lower threshold for regular use? Notably, this cut-off excluded the Gates study, which included data on women who used talc more

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58 Siemiatycki Rep. at 41.

59 *Id.* at 31.

60 *Id.* at 30.

61 *Id.* at 32.

62 *Id.* at 34 ("I tried to be consistent in defining exposure, but this factor was subjectively determined by the individual studies."); *see* Smith-Bindman Dep. Vol. II 272:3-273:3 (Smith-Bindman "tried to approximate regular use" and has not validated her metric).

63 Smith-Bindman Rep. at 32.

64 *Id.*

65 *Id.*

than once per week (as explained below, including Gates in the meta-analysis would have lowered the odds ratio Dr. Smith-Bindman calculated for serous invasive). What criteria governed her determination of whether a study's description "made it clear" that it really addressed regular use? Why did she only use the highest use category when studies reported multiple categories of regular use, and what made such a group "large enough to be meaningful"? How did she determine "which data may have reflected daily use"? Although Dr. Smith-Bindman speculated at her deposition that other epidemiologists could repeat her analysis using her methodology,[66] the lack of a clear protocol and the need to reproduce seemingly arbitrary decisions in her assessment of "regular" talc use would make that very difficult, if not impossible. And importantly, whether a study reported on "regular" talc use appears to be the sole criterion Dr. Smith-Bindman employed in choosing studies for her review.

- Dr. Smith-Bindman excluded studies that examined talc use on sanitary napkins, diaphragms or condoms, claiming (without any supporting data) that perineal use is "the most common exposure type and is likely to reflect the most consistent exposure."[67] Notably, if the talc migration theory Dr. Smith-Bindman endorses is correct, data regarding talc use on condoms and diaphragms should be especially valuable, since such use introduces talc directly into the vagina. But studies examining such use have not reported an association with ovarian cancer, and her decision to exclude them again illustrates that she systematically avoided data that did not support her desired result.

- Even after designing a study selection methodology that enabled her to cherry-pick studies that supported finding an association, Dr. Smith-Bindman omitted Rosenblatt 2011 after initially selecting it.[68] That study reported negative associations between talc use and both invasive serous ovarian cancer and ovarian cancer overall for its two highest use categories.[69] At her deposition, Dr. Smith-Bindman speculated that she omitted this study because doing so did not make a difference in her results.[70] That is both an odd reason to exclude a study (all other things being equal, a more robust data set is obviously preferable) and objectively wrong, since her underlying data show that omitting the Rosenblatt study increased her odds ratio for frequent use and serous invasive from 1.38 to 1.52 and for frequent use and all ovarian cancer from 1.32 to 1.43.[71] In other words, if Dr. Smith-Bindman had not excluded Rosenblatt 2011 from her final results, she would

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66 Smith-Bindman Dep. Vol. II 357:1-15; Smith-Bindman Dep. Vol. I 102:21-104:18; *see, e.g.*, Smith-Bindman Dep. Vol. I 197:19-198:6 (Smith-Bindman has no written protocol setting forth the myriad assumptions she and her colleague made when abstracting data).

67 Smith-Bindman Rep. at 32-33.

68 *Id.* at 33-34.

69 Rosenblatt 2011 at Table 2 (reporting point estimates of 0.78 and 0.87 for invasive tumors and all tumors in women with between 4,800 and 9,999 lifetime applications; and 0.84 and 0.87, respectively, for women with more than 10,000 lifetime applications).

70 Smith-Bindman Dep. Vol. I 177:20-180:13.

71 TalcDataResults-janehall.xlsx (compare "All papers" tab with "Excluding Rosenblatt" tab).

not have been able to opine that regular talc use is associated with a 50 percent increase in the risk of serous invasive ovarian cancer.[72]

- The pool of studies on which she relies for her assessment of serous invasive ovarian cancer is very small – consisting of only four reports and far fewer cases overall than the broader pool of ovarian cancer studies, making the dataset less robust. Moreover, while Dr. Smith-Bindman includes the Gertig study as one of the four in her consideration of serous ovarian cancer risk, she omits the Gates study,[73] which, as noted above, updated the findings of the NHS on which Gertig had reported and concluded after ten more years of study that there was no association between talc use and serous ovarian cancer specifically (RR 1.06 (95% CI: 0.84-1.35)).[74] Had Gates been considered, Dr. Smith-Bindman's reported overall odds ratio of 1.52 for serous ovarian cancer would presumably have been much lower.

- As noted above, Dr. Smith-Bindman admittedly made no effort to assess the quality of the case-control studies that comprise 90 percent of her meta-analysis.[75] As she confirmed at her deposition, this included no effort to assess whether the studies she selected adequately controlled for bias and confounding.[76]

- Dr. Smith-Bindman admittedly abstracted inaccurate data from the studies she considered in her review.[77] Indeed, none of the confidence interval data she reports for the ten studies she includes in Figures 2 and 3 of her report match what was reported in the studies themselves.[78] This is surprising since Dr. Smith-Bindman acknowledges that data abstraction is an extremely important step for a meta-analysis and that having inaccurate

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72 *See* Smith-Bindman Rep. at 34.

73 *Id.*

74 Gates 2010 at 50.

75 Smith-Bindman Rep. at 29.

76 Smith-Bindman Dep. Vol. II 311:18-312:24; *see id.* 284:7-15 (agreeing that bias in underlying studies does not disappear when they are combined in a meta-analysis).

77 Smith-Bindman Dep. Vol. I 105:14-21.

78 *Id.* 182:13-183:24. *Compare Smith-Bindman* Rep. at 33 fig. 2, *with* Booth 1989 at 596 (Smith-Bindman (0.75-1.85) vs. study (0.8-1.9)), *and* Chang & Risch 1997 at 2399 (Smith-Bindman (0.51-1.39) vs. study (0.61-1.49)), *and* Cook 1997 at 462 (Smith-Bindman (0.55-3.05) vs. study (1.2-2.9)), *and* Cramer 2016 at 335 (Smith-Bindman (0.97-2.01) vs. study (1.06-2.10)), *and* Gertig 2000 at 250 (Smith-Bindman (0.76-1.48) vs. study (0.82-1.55)), *and* Harlow 1992 at 19 (Smith-Bindman (0.85-2.75) vs. study (1.1-3.0)), *and* Mills 2004 at 460 (Smith-Bindman (0.93-2.55) vs. study (1.14-2.64)), *and* Schildkraut 2016 at 1413 (Smith-Bindman (1.18-2.24) vs. study (1.26-2.33)), *and* Whittemore 1988 at 1231 (Smith-Bindman (0.81-2.09) vs. study (0.81-2.60)), *and* Wu 2009 at 1409 (Smith-Bindman (1.14-3.02) vs. study (1.34-3.23)). *Compare* Smith-Bindman Rep. at 34 fig. 3, *with* Chang & Risch 1997 at 2399 (Smith-Bindman (1.07-1.96) vs. study (1.13-2.02)), *and* Cook 1997 at 462 (Smith-Bindman (0.55-3.05) vs. study (1.2-2.9)), *and* Cramer 2016 at 342 (Smith-Bindman (1.08-2.00) vs. study (1.15-2.07)), *and* Gertig 2000 at 250 (Smith-Bindman (0.86-2.12) vs. study (0.98-2.26)).

data can compromise a meta-analysis.[79] Dr. Smith-Bindman also admits that she likely double-counted patients in her data, despite acknowledging that this should be avoided.[80]

In short, Dr. Smith-Bindman's meta-analysis is arbitrary, error-laden and designed to systematically exclude data that do not support the theory that talc use causes ovarian cancer. These methods are unreliable. Based on her meta-analysis, Dr. Smith-Bindman concluded that she does "not have ***any uncertainty*** that regular exposure to talc powder products" increases the risk of ovarian cancer;[81] yet, it is difficult to conceive how use of such a methodology would not introduce ***substantial uncertainty*** into a meta-analysis, as well as any interpretation of its results.

2. Bias

Bias is a particularly important issue when analyzing whether perineal exposure to talcum powder causes ovarian cancer because, as set forth above, the reported risks are very small. The reporting of small risks suggests these studies are susceptible to biases.[82]

Additionally, case-control studies are particularly susceptible to bias (although I agree with Dr. McTiernan that hospital-based studies may be less distorted by recall bias than population-based studies because the former feature both ill cases and ill controls).[83] In most of the case-control studies pertaining to perineal talcum powder use and ovarian cancer, the authors discuss the potential for bias, including recall bias. However, only one study examined the issue directly, and it found striking and clear evidence of the impact of recall bias on the study results.

In the case-control study reported by Schildkraut et. al., the authors (including Dr. Moorman) considered that "the possibility of differential misclassification exists in a case-control study such as AACES, especially due to the heightened awareness of the exposure as a result of" well-publicized litigation.[84] The investigators examined their finding based on whether the study subjects were interviewed before 2014 versus 2014 onward. Among those interviewed before 2014, the reported use of body powder on the genitals was nearly the same for cases and controls (36.5 and 34.0%, respectively). But from 2014 onward, the reported use among cases was markedly higher (51.5%), while it stayed the same in controls (34.4%). This striking and abrupt change in reporting clearly demonstrates the major impact of recall bias, and that plaintiffs' experts are wrong to label recall bias in case-control studies "theoretical."[85] But it also calls into question earlier results because – contrary to Dr. Moorman's claim that "the vast

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79 Smith-Bindman Dep. Vol. I 104:22-105:13, 106:6-13; Smith-Bindman Dep. Vol. II 282:16-283:3.

80 Smith-Bindman Rep. at 34; Smith-Bindman Dep. Vol. II 344:9-345:3.

81 Smith-Bindman Rep. at 4 (emphasis added).

82 Moorman Dep. 251:2-7 ("I think that with a smaller association, there is more concern that it could be due to bias from various reasons.").

83 McTiernan Rep. at 24

84 Schildkraut 2016 at 1416.

85 McTiernan Rep. at 20; Moorman Rep. at 23.

majority of studies" were not affected by this issue[86] – the question of talc and ovarian cancer did not emerge for the first time in 2014, and earlier studies could well have been affected by a more modest but nonetheless significant recall bias. Clearly, media reporting about talc and ovarian cancer did not begin in 2014; rather, there are multiple news reports between 1982 and 2013 (**See Appendix A: Sample Of Pre-2014 News Articles Addressing Posited Link Between Talc Use And Ovarian Cancer for a list of examples**). Women with ovarian cancer in that era could easily have been influenced in their recall of talcum powder use, which would potentially amplify recall bias in pre-2014 studies as well.

Dr. Moorman further argues that "empirical evidence" shows that recall bias in case-control studies is only a theoretical concern, citing a study by Lanza et al. that found that case-control and cohort studies reached similar results regarding certain therapeutic interventions.[87] But the Lanza findings (which had nothing to do with talc) are obviously not applicable to the situation here, where the case-control and cohort studies at issue have been highly heterogeneous.[88]

Other study authors have recognized the problem of bias in their studies as well. For example, a 2017 pooled study of 12 case-control studies addressing ovarian cancer risk factors in four ethnic groups found a statistically significant elevated risk for talc use among two of the four ethnic groups (Non-Hispanic White (OR 1.30 (95% CI: 1.20–1.41)) and Black (OR 1.62 (95% CI: 1.32–2.00)) and no statistically significant elevated risk for the other two groups (Hispanic (OR 1.41 (95% CI: 0.93–2.13)) and Asian/Pacific Islander (OR 1.02 (95% CI: 0.61–1.70))).[89] The authors characterized the differences across groups as "[s]tudy heterogeneity" and cautioned: "A concern with self-reported data is recall bias, especially for characteristics that are difficult to report with accuracy, require subjective summarization or can be influenced by the investigator, media or similar factors. Such problematic characteristics may include body powder exposure[.]"[90]

3. Confounding

Similarly, confounding factors may have affected the studies that found a small estimated risk pertaining to perineal exposure to talcum powder and ovarian cancer. This issue is especially concerning when it comes to ovarian cancer risk because, generally, scientists do not know the cause of ovarian cancer.[91] Thus, even studies that attempt to account for known confounders (such as familial or genetic risk) likely do not account for most of the risks – known or unknown.

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86 Moorman Rep. at 23.

87 Moorman Rep. at 23; Moorman Dep. 227:11-23.

88 *See* Moorman Dep. 227:24-232:15.

89 Peres LC, Risch H, Terry KL, et al. Racial/ethnic differences in the epidemiology of ovarian cancer: a pooled analysis of 12 case-control studies. Int J Epidemiol. 2018; 47(2):460-472.

90 *Id.* at 8-9.

91 Siemiatycki Dep. 173:6-9 (agreeing that "all of the factors that might make someone susceptible to developing ovarian cancer are not currently known").

The Sister Study[92] provides insight into one potential source of confounding in prior studies. In that study, the investigators accounted for douching, an exposure not considered in nearly all other studies. The authors were interested in douching because of concerns that it could "introduce particles and toxicants in the upper reproductive tract and increase the risk of cancers and infections." They cited evidence that douching products contain phthalates that "could influence ovarian cancer risk through hormone disruption." The study found that douching was a risk factor for ovarian cancer (HR 1.8 (1.2-2.8)), while talc use was not (HR 0.73 (0.44-1.2)). Douching, with or without concurrent talc use, had similar risk (HR 1.8 and 1.9, respectively). The investigators noted that the practice of douching and talc use are correlated and that "if douching is a risk factor for ovarian cancer, some of the earlier reports on talc could have been subject to confounding bias." The same study also showed that douche users are different from non-users, with users more likely being Non-Hispanic Black, of lower educational attainment and/or obese. These systematic differences highlight the complexity of understanding the potential effect of a non-random feminine hygiene practice and judging causation when estimated risks are otherwise so small.

The finding in Gonzalez that the douche users had lower educational attainment suggests that socioeconomic status may be another important confounder. Indeed, in another study by Alberg et al. the investigators found that higher educational attainment may be protective against developing ovarian cancer (or in other words, low educational attainment is associated with higher risk of developing ovarian cancer).[93] The authors noted that if socioeconomic status is truly protective, the reasons for the relationship still need to be identified.[94] They suggested that differences in diet and exercise could be related to risk, which overall means that assessing confounding in ovarian cancer studies is important, complex and not yet fully developed in research.[95] What is important in assessing the epidemiologic studies of talc and ovarian cancer is that, as Dr. Smith-Bindman acknowledges, the studies did not use a uniform approach to assessing confounders, with, for example, nearly all not adjusting for douching and many not accounting for education or socioeconomic status.[96] Accordingly, Dr. McTiernan's argument that confounding is unlikely because studies have reported small differences between adjusted and crude results is overly simplistic (and in any event ignores that studies cannot adjust for unknown confounders).[97]

4. Other Considerations

It is important to recognize that the strength of an association is not the same as the importance of the association. The importance of an association is based on the judgment of

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92 Gonzalez 2016.

93 Alberg AJ, Moorman PG, Crankshaw S, et al. Socioeconomic Status in Relation to the Risk of Ovarian Cancer in African-American Women: A Population-Based Case-Control Study. Am J Epidemiol. 2016; 184(4):274-283.

94 *Id.* at 282.

95 *Id.*

96 Smith-Bindman Dep. Vol. II 307:21-308:24.

97 McTiernan Rep. at 24; McTiernan Dep. 176:17-177:23.

those using the information. A new medication that reduces death from heart attacks by 2% may be judged to be very important, and if that drug causes itching in 30% of users, that finding may be judged less important. An effect that is judged to be important is not evidence of causation, however.

In this matter, some of plaintiffs' experts have provided confusing opinions about strength of association. While the strength of association between talcum powder use and ovarian cancer is indisputably small, the experts have nevertheless found it to be "strong" by discussing their judgments about the potential importance of the findings and also by bringing in other arguments, such as statistical significance. For example, Dr. Smith-Bindman states:

> "It is frequently argued that the larger an apparent association, the more likely the association is to be real (causal) and important for epidemiological assessment. This would suggest that an OR of 2.0 is more likely to indicate causality and importance than an OR of 1.5. While this is often argued, I do not believe this is necessarily the case. If a risk factor increases the risk of disease by 50%, and the exposure is common, it will have far greater impact on a number of people, in comparison to a rare exposure that has a higher associated OR. A larger association between exposure and disease may be easier to identify, but I do not believe it is more likely to indicate causality or importance."[98]

Dr. Smith-Bindman is conflating the distinct issues of causation and importance in arguing that "the data supporting the causality of talcum powder products exposure for ovarian cancer is extremely strong."[99] She goes on to calculate the number of ovarian cancers she believes are caused by talcum powder products and uses this calculated number of cancers to support her statement that "this Bradford Hill Factor of the Strength of the association is important and met."[100] In other words, Dr. Smith-Bindman opines that because, according to her calculations, a large percentage of ovarian cancer is caused by talcum powder, the association is "strong." This statement is misleading and circular because Dr. Smith-Bindman is using the "importance" of the finding, which is only important if true (i.e., causal), to support the judgment that the very small association is causal. One needs to first determine if an association is causal, and only then, if it is causal, decide on its importance.

Other plaintiffs' experts make similar conflated arguments. Dr. Blair Smith uses the potential importance of the finding in her assessment of the strength of association when she states that "there is no set magnitude or threshold for ascribing causality. I would maintain that any practice or element that increases the risk of ovarian cancer by ANY consistent percentage is significant."[101] Dr. Moorman also states that it is "critical to consider the prevalence of the exposure" in assessing strength of association and "how many cases of disease could be

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98 Smith-Bindman Rep. at 36.

99 *Id.* at 37.

100 *Id.* at 38.

101 Blair Smith Rep. at 19.

attributable to this exposure."[102] Likewise, Dr. McTiernan, although admitting the risks are approximately 22-31% (equivalent to RR ~1.2-1.3), expressed the opinion that the association is strong because "given the high prevalence of use of talcum powder products" in this population, these levels of risk present a clinically significant public health concern."[103] Thus, Drs. Blair Smith and McTiernan are using the concept of importance to justify the strength of a very small numerical risk. Furthermore, Dr. McTiernan's opinion about the strength of association is confusing for the additional reason that she folds in other criteria such as consistency of findings, which should be assessed separately. Dr. McTiernan's conflation of many different concepts makes her Bradford Hill analysis unreliable.

Plaintiffs' experts also cite to examples of "established carcinogens" with similar estimates of strength of association – like passive smoke exposure and lung cancer, or hormone replacement therapy and breast cancer – to conclude that the association between talc and ovarian cancer is strong enough to be causative.[104] Although it is true that the associations are numerically similar, it is improper to conclude that any association of the same size is causal. After all, for those other exposures, the fact of a weak association may have been overcome by strong evidence that the other Bradford Hill criteria were met. And, as Dr. Moorman concedes, there are also examples of numerically similar associations that have not been established as causal.[105] Additionally, causation for certain of these other examples was based on data from randomized trials, which are the strongest evidence of a causal relationship. For example, the clinical trials pertaining to hormone therapy and breast cancer randomly assigned patients to treatment and control groups, rendering a high likelihood that any association that is observed is due to the exposure, as opposed to bias or confounders. In other words, the causal relationship between hormone therapy and breast cancer is based on better data, not on the finding of a small association.[106]

Additionally, plaintiffs' experts' heavy reliance – and in the case of Dr. McTiernan, exclusive reliance[107] – on meta-analyses and pooled analyses to demonstrate strength of association is flawed in many respects. First, as plaintiffs' experts have repeatedly acknowledged, the meta-analyses do not eliminate the bias inherent in the underlying studies.[108] And although plaintiffs' experts focus on newer studies,[109] Dr. Siemiatycki admits that the

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102 Moorman Dep. 261:1-262:1.

103 McTiernan Rep. at 9.

104 Singh Rep. at 17; Moorman Rep. at 12; Moorman Dep. 245:10-16; Siemiatycki Dep. 148:8-19.

105 Moorman Dep. 255:12-25 ("I acknowledge that – of course, that there are reports of exposures that have reported relative risk in this range, and it could either be something that was associated with another risk factor and it was not the causal factor or the level of evidence was not adequate.").

106 Chlebowski RT, Hendrix SL, Langer RD, et al. Influence of estrogen plus progestin on breast cancer and mammography in healthy postmenopausal women: the Women's Health Initiative Randomized Trial. JAMA. 2003; 289(24):3243-3253.

107 McTiernan Rep. at 63; McTiernan Dep. 243:7-14.

108 McTiernan Dep. 244:9-13; Moorman Dep. 159:8-160:18.

109 McTiernan Dep. 282:2-4.

relative risks have gone down as more data has been collected over the years. For example, the 1.28 odds ratio provided by Dr. Siemiatycki in his 2018 meta-analysis is lower than the 1.35 relative risk published in the 2008 Langseth article.[110] And Dr. Siemiatycki acknowledged that the Berge 2018 authors noted a downward trend in the risk assessment over time.[111]

Finally, Dr. Smith-Bindman reports finding a slightly higher odds ratio of 1.52 by focusing on the particular histologic subtype of serous ovarian cancer.[112] But this alternative approach to the issue of strength does not materially affect the analysis. An odds ratio of 1.52 remains well below 2.0 and would still be considered a weak association. The studies offering odds ratios for serous ovarian cancer, like the broader pool of studies, contain a mix of findings, with some reporting statistically significant findings and others not.[113] And Dr. Smith-Bindman's methodology to reach the 1.52 odds ratio was deficient for the numerous reasons discussed above.

Based on the foregoing, it is my opinion that the association between perineal talcum powder exposure and ovarian cancer is weak and likely impacted by bias, confounding and/or chance. Moreover, plaintiffs' experts' attempts to explain away these problems and cast the science as standing for essentially the opposite proposition – that the epidemiology establishes a strong or conclusive association – strongly suggest that they are engaged in advocacy rather than science.

B. Epidemiologic Studies Are Inconsistent.

As set forth above, the prospective epidemiologic studies (cohort studies) do not show a statistically significant association, while only a subset of the population-based case-control studies does.[114] This disparity reflects inconsistent results across different types of studies, undermining the conclusion that cosmetic talc use causes ovarian cancer. The fact that none of the cohort studies found a statistically significant association between talc use and ovarian cancer is critical in this regard,[115] because it calls into doubt even the modest association in some of the population-based case-control studies.

Other inconsistencies exist in the literature as well, including some that overlap with the concepts of coherence and plausibility.[116] Evaluating an association with the use of talc-dusted

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110 Siemiatycki Dep. 149:14-150:3.

111 Siemiatycki Dep. 206:21-207:19.

112 Smith-Bindman Rep. at 34.

113 *Id.* at 34 fig. 3 (citing four studies, one of which (Cook 1997) reported a statistically insignificant result, and two of which have confidence intervals that are only barely above 1.0).

114 As just explained, this disparity holds for the subtype of serous ovarian cancer as well, as to which the Gates study reported no statistically significant association.

115 Berge 2018 at 251.

116 Fiume MM, Boyer I, Bergfeld WF, et al. Safety Assessment of Talc as Used in Cosmetics. Int J Toxicol. 2015; 34(1 Suppl):66S-129S, 119S ("Fiume 2015"); Hartge 1983; Muscat JE, Huncharek MS. Perineal talc use and

diaphragms and condoms has been deemed "the most valid method for testing the carcinogenic potential of talc" because "[b]y definition, the female reproductive tract is exposed to talc containing powders introduced by diaphragms, whereas an exposure route based on perineal dusting requires unproven assumptions about vaginal exposure."[117] Studies pertaining to use of talcum powder on diaphragms and condoms have shown a consistent lack of risk. It is illogical that talcum powder applied to the outside of the genital tract can cause ovarian cancer, while talcum powder applied inside the genital tract would not. Additionally, assertions by plaintiffs' experts that these studies are "obsolete" due to a "lower methodological quality"[118] are merely unfounded assertions.

Some of plaintiffs' experts still argue that the data on the association between genital talc use and ovarian cancer are highly consistent, but their explanations fail.

For example, Dr. McTiernan states that "[a]cross the case-control and cohort studies, the association between use of talcum powder products and risk of ovarian cancer was highly consistent."[119] This statement is simply not true: while some of the case-control studies have shown a small positive risk, the cohort studies have uniformly failed to demonstrate a risk, as Dr. McTiernan admits.[120] She states further that because the cohort studies "were not well designed to determine true risk for ovarian cancer and perineal talc use their results as a group do not negate the significant case-control findings."[121] But her criticisms of cohort studies are misplaced, as previously discussed. In any event, her argument assumes that the results of some studies are not consistent, or else there would be no reason for Dr. McTiernan to find fault with the cohort study designs in order to explain why their results do not negate the findings from other studies. Furthermore, Dr. McTiernan completely ignores the fact that within the case-control studies, there is evidence of inconsistency based on the type of control group. The different findings in the case-control studies by type of control group is further evidence of inconsistency.

Drs. Singh and Moorman purport to find consistency because "[t]he meta-analysis of case-control studies has consistently shown a statistically significant increased risk" and "the meta-analysis of cohort studies has also shown an excess risk, [] which failed to reach statistical significance."[122] This consistency analysis is faulty for two reasons. First, since meta-analyses

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ovarian cancer: a critical review. Eur J Cancer Prev. 2008; 17(2):139-146, 144-145 (2008) ("Muscat & Huncharek 2008").

[117] Muscat & Huncharek 2008.

[118] Singh Rep. at 17, 26-27.

[119] McTiernan Rep. at 64.

[120] McTiernan Dep. 200:25-201:10 ("So yes, there was heterogeneity between the case-control and cohort studies"), 202:17-203:1 ("I agree that the cohort studies have lower relative risks than do the case-control studies, yes.").

[121] McTiernan Rep. at 64.

[122] Singh Rep. at 17; Singh Dep. 146:25-147:5 (stating "[t]he cohort studies show . . . increased risk, which is in the same direction as the case-control studies"); Moorman Dep. 262:20-264:13 (explaining that "both the Houghton study and the Nurses' Health Study . . . are consistent in terms of the direction of the effect").

analyze overlapping sets of individual studies, it is not surprising that meta-analyses yield consistent results. For this reason, consistency as determined by the meta-analyses' estimates is not supportive of Bradford Hill's consistency of association criterion. Second, as Drs. Singh and Moorman admit, the purported "excess risk" or "direction of the effect" shown in the meta-analyses of cohort studies does not amount to "statistical significance."[123] Drs. Singh and Moorman's classification of "excess risk" or "direction of the effect" glosses over the fact that case-control studies and cohort studies found varying strengths of association that do not amount to consistent results. Similarly, Dr. McTiernan's purported assessment of consistency by "look[ing] at whether the relative risk is above one consistently" is so broad that it is nonsensical, as it would consider near-null associations and definitively causal associations consistent.[124] And plaintiffs' experts' argument that certain studies would have shown a statistically significant increased risk if they had larger sample sizes (i.e., Dr. McTiernan with respect to cohort studies and Dr. Moorman with respect to small, non-statistically significant associations in African American and white women found in her 2009 study)[125] is speculative because there is no way to know whether a larger sample would provide the same or a different estimate or whether that estimate would be statistically significant. I note that the fact that Dr. Moorman did not include these results from her own 2009 study in her report suggests a biased approach to synthesizing the literature.[126] In any event, Drs. McTiernan and Moorman likewise ignored the Berge study's analysis demonstrating that the cohort studies collectively had sufficient power to detect a 1.25 relative risk if one existed; as the authors stated, "low power of cohort studies cannot be invoked as [an] explanation of the heterogeneity of results."[127]

C. Specificity Is Not Compelling.

Specificity was not considered very important by plaintiffs' experts and I agree.[128] There is no compelling case for specificity here either.

D. The Epidemiological Data Do Not Show Biological Gradient (Dose Response).

1. Available Epidemiological Data On Dose-Response

Evidence of dose-response – i.e., whether the risk of developing ovarian cancer increases with increased perineal talc exposure – is one of the most important factors to consider in

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123 Moorman Dep. 266:6-16.

124 McTiernan Dep. 212:17-21; *see* McTiernan Rep. at 44 (considering the near-null and not statistically significant 1.06 odds ratio reported in Houghton 2014 evidence of consistency of association); Moorman Dep. 263:13-264:13 (similar).

125 McTiernan Rep. at 45-46; Moorman Dep. 136:12-19.

126 Moorman Dep. 136:21-137:2; *see* Moorman PG1, Palmieri RT, Akushevich L, et al. Ovarian cancer risk factors in African-American and white women. Am J Epidemiol. 2009; 170(5):598-606 (reporting non-statistically significant odds ratios of 1.19 and 1.04 for African American and white women, respectively).

127 Berge 2018 at 253; *see* Moorman Dep. 213:2-23; McTiernan Rep. at 46-47.

128 Singh Rep. at 64.

evaluating causation. The epidemiological literature studying talc and ovarian cancer has failed to show a dose-response relationship. Plaintiffs' experts claim that there is sufficient data supporting the existence of a dose-response relationship[129] and have pointed to some studies as purported evidence of dose-response, including, for example, the articles by Schildkraut and Cramer.[130] But overall, the literature is very inconsistent with regard to dose-response, as Drs. Smith-Bindman and Moorman concede.[131]

None of the cohort studies (Gonzalez 2016; Houghton 2014; and Gates 2010/Gertig 2000) demonstrates a dose-response relationship, and only a handful of case-control studies (Harlow et al. 1992; Cramer 2016 and Schildkraut 2016) have purported to find one. The case-control studies have in fact shown a wide variety of findings, including: (1) a positive dose-response; (2) no dose-response; (3) a negative dose-response; and (4) a haphazard or bizarre pattern. Notably, among the numerous case-control studies that have not reported a dose-response relationship are several studies that have analyzed "cumulative" talc use (otherwise known as "frequency times duration" of use). For example, Mills 2004 examined cumulative dose by quartiles and reported risks of 1.03, 1.81, 1.74 and 1.06 for ascending quartiles – a bizarre trend that does not support there being a dose response.[132] Similarly, the Cook 1997 study looked for an association across various strata of "cumulative lifetime days."[133] The results showed no statistically significant elevated risk for any of the four categories, with the relative risk for the lowest group (fewer than 2,000 cumulative days, RR 1.8 (95% CI: 0.9-3.5)) essentially matching that of the highest group (greater than 10,000 cumulative days, RR 1.8 (95% CI: 0.9-3.4)).[134] Moreover, as noted above, the Rosenblatt 2011 study looked at the association across four categories of increasing lifetime applications and reported the lowest associations (in fact, negative associations) for its two highest use categories.[135] In addition, Chang found an inverse relationship with risks related to use per month of 1.8, 1.1 and 0.9 for respectively <10, 10-25 and more than 25 applications; similar inverse findings for years of use were 1.7, 1.4 and 0.86 for <30, 30-40 and >40 years of use.[136]

Although some studies have purported to observe a dose trend with cumulative use, those results are not meaningful. For example, the Schildkraut study only compared women who had used talc fewer than 20 years versus more than 20 years and fewer than 3600 applications versus

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129 Singh Rep. at 55-56; Smith-Bindman Rep. at 39-40.

130 Singh Rep. at 56.

131 Smith-Bindman Rep. at 40 ("most but not all studies of talcum powder products and ovarian cancer show a dose response, but the results are inconsistent, and more importantly, are not considered or assessed in most of the published studies"); Moorman Dep. 272:5-10 ("across the studies, some have found a dose-response, some have not").

132 Mills 2004.

133 Cook 1997 at 463.

134 *Id.*

135 Rosenblatt 2011 at 740.

136 Chang & Risch 1997.

more than 3600 applications.[137] Although it found statistically significant associations for the higher but not lower use categories, the study provides little useful information about dose-response because exposure is crudely dichotomized into just two categories each for frequency and duration. And the Cramer study found essentially no difference – and certainly no steady increase – in risk as to women who had (as the study calculated) used talc for the equivalent of 1-5 years, 5-20 years and more than 20 years (odds ratios of 1.36, 1.41 and 1.39, respectively).[138]

Several meta-analyses and pooled studies have analyzed the body of studies and resoundingly concluded that there is not a demonstrated dose response. For example, the 2013 Terry pooled study of eight case-control studies addressed the potential association between ovarian cancer and the use of powder (broadly defined to include both talc and cornstarch).[139] One of the primary goals of the analysis was to determine whether a dose-response relationship existed, as previous evidence "ha[d] been inconsistent."[140] The authors found that it did not.[141] Indeed, although Dr. Siemiatycki claims that this study is "the most important evidence around dose-response," the authors stated that they "observed ***no significant trend in risk with increasing number of lifetime applications***," as he has acknowledged.[142] The Terry study, in fact, only observed a positive dose trend when including non-talc users in the analysis,[143] which is not actually meaningful evidence of a dose response, since including nonusers in a dose-response analysis makes that analysis redundant with whether there is an association with ever/never use, as Dr. Siemiatycki acknowledges.[144] Although Dr. Siemiatycki argues that it may be appropriate to include nonusers in the dose-response analysis when a study only reports on dose-response and not ever/never use,[145] that clearly does not apply to the Terry study, which reported on both types of data. Of note, the Terry authors did not mention the trend with nonusers in their abstract or discussion, instead highlighting that they found "no significant [dose] trend" and explaining that "[w]hether risk increases with number of genital powder applications and for all histologic types of ovarian cancer . . . remains uncertain."[146] The authors also acknowledged that, if anything, the study might ***overstate*** the relationship between powder use and ovarian cancer if cases [i.e., women with ovarian cancer] were more likely to report

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137 Schildkraut 2016 at 1415-1416 (Table 2).

138 Cramer 2016 at 337 (Table 1).

139 Terry 2013 at 812.

140 *Id.*

141 *Id.*

142 *Id.* at 811, 812 (emphasis added); Siemiatycki Dep. 197:17-22, 266:8-15, 268:14-21.

143 Terry 2013 at 817.

144 Siemiatycki Rep. at 43 ("If the Ever/Never result is presented and then a dose-response analysis is conducted, it is preferable to maintain statistical independence of the two analyses by excluding the baseline unexposed category from the dose-response analyses).

145 *Id.*

146 Terry 2013 at 811, 819-20.

genital-powder use than controls [i.e., women without ovarian cancer]."[147]

Similarly, a 2008 meta-analysis identified "the absence of clear exposure-response associations in most studies" as a crucial piece of missing evidence needed to establish causation.[148] And in assessing the body of literature, the National Cancer Institute ("NCI") and the United States Food & Drug Administration ("FDA") have respectively concluded that "a dose response relationship was not found" and that "dose-response evidence is lacking."[149] Although two more recent meta-analyses claimed to find evidence of a very small dose-response, these data are not compelling. Specifically, Berge 2018 reported a "weak" dose-response trend, but cautioned that these data came from a small number of case-control studies.[150] And Penninkilampi divided talc users into only two categories (greater and fewer than 3,600 lifetime applications), finding only a "slightly greater increased risk" for the former category (also based only on case-control data).[151] As with Schildkraut, the arbitrary dichotomous categorization of lifetime use further undercuts the significance of this finding.

Consistent with these results, pathological studies have not reported a correlation between the amount of talc used and talc particle counts in ovaries. As one study explained: "ovarian talc particle burden has been found not to correlate with the reported number of lifetime applications, which (if not reflective of inaccurate reporting) may indicate that duration of the powder use is not relevant when assessing risk associated with differing levels of exposure to talc."[152]

In sum, the findings of so many different patterns, or lack of patterns, by dose-response estimation weighs against causation, and indeed, the fact that the data show no clear dose trend is consistent with there being no causal relationship. If one were to believe that perineal talcum powder use causes ovarian cancer, these mixed and inconsistent results should cast serious doubt

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147 *Id.* at 820.

148 Langseth 2008 at 359; *see* Gertig 2000 at 249, 251 (cohort study concluding that "[w]e did not observe a dose-response relationship with talc use, and previous studies have been inconsistent in this regard"); Cramer 1999 at 355 (case-control study by Dr. Daniel Cramer conceding that "[t]he most obvious weakness in the argument for biologic credibility of the talc and ovarian cancer association is the lack of a clear dose response" and that "[m]ost talc and ovarian cancer studies that have addressed dose response, including this one, have failed to demonstrate consistent dose response relationships").

149 National Cancer Institute, Ovarian, Fallopian Tube, and Primary Peritoneal Cancer Prevention (PDQ®)–Health Professional Version, https://www.cancer.gov/types/ovarian/hp/ovarian-prevention-pdq (last updated Jan. 4, 2019); Letter from Food & Drug Administration, U.S. Department of Health and Human Services, to Samuel S. Epstein, M.D., Cancer Prevention Coalition, University of Illinois (Apr. 1, 2014);International Agency for Research on Cancer, Monographs on the Evaluation of Carcinogenic Risks to Humans Vol. 93: Carbon Black, Titanium Dioxide, and Talc 18-19 (2010) ("IARC Talc Monographs") (concluding that evidence of a dose-response relationship was "inconsistent").

150 Berge 2018 at abstract, 255.

151 Penninkilampi 2018 at 45.

152 Rosenblatt 2011 at 742 (discussing Heller DS, Westhoff C, Gordon RE, Katz N. The Relationship Between Perineal Cosmetic Talc Usage and Ovarian Talc Particle Burden. Am J Obstet Gynecol. 1996; 174(5):1507-1510 ("Heller 1996")).

on the validity of the measures used to estimate whether and how much talcum powder was used.

2. Validity Of Exposure Measure

In epidemiologic research, it is critical to assess exposures of interest with accuracy and precision. This includes measuring exposures with tools that have demonstrable validity. Without a validated measure of exposure, it is not possible to know whether or not an exposure occurred, and even if it did, it is not possible to quantify the exposure with any degree of certainty.

While there is a scientific approach for development and testing of survey questions for use in research,[153,154,155] there is not a single epidemiologic study of the potential association between perineal use of cosmetic talcum powder and ovarian cancer that has used, or purports to have used, a validated measure of talcum powder use.[156] Thus, it is unknown whether any of the studies have accurately assessed whether talcum powder was used, for how long and how frequently. Self-report measures can be highly inaccurate, and none has been shown to be valid. In studies of medication use, for example, validation of self-report can come from examining pharmacy dispensation records or deploying electronic counters to medications as objective measures to validate a person's reported use of a medication. No such efforts have gone into the development of questions about self-reported talc use.

Even more important, perhaps, is that no study has a measure that has been shown to estimate the relevant dose of talcum powder. An "application" of talcum powder has no standard definition. It is unknown how much, if any, talcum powder reported on any of the questionnaires is applied to the perineum, how much, if any, reached the vagina, nor how much, if any, reached the ovaries. The problem is especially profound in this context, because slight inaccuracies in estimating the amount used on a daily basis could significantly alter total estimated use where the history in some cases spans multiple decades. Thus, it is impossible from the studies to determine how much, if any, talcum powder was applied to the perineum, and likewise impossible to measure how much, if any, talcum powder migrated into the vagina, across the cervix, up through the uterus and eventually reached the ovaries. At best, the wide variety of non-validated measures of talcum powder use can collect hypothesis-generating data, and there is no assurance that any estimates of talc use are accurate or valid.

Below is an example of the challenges presented when a validated measure of exposure is nonexistent:

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153 *See generally* Aday LA, Cornelius LJ. Designing and Conducting Health Surveys: A Comprehensive Guide. 3rd ed. San Francisco, CA: 2006.

154 Fowler FJ Jr. Survey Research Methods. 5th ed. Thousand Oaks, CA: 2014.

155 Seifert B. Validity criteria for exposure assessment methods. Sci Total Environ. 1995; 168(2):101-107.

156 *See* McTiernan Dep. 53:18-22 ("It was not possible to determine exactly how much talcum powder product was used").

- Consider the question of whether or not consumption of milk can either cause or protect against the development of allergies. It might seem simple that one could design a survey to ask people, with and without allergies, about their past consumption of milk. A question could be: in the past 12 months, did you drink milk? People with and without allergies could be compared by whether or not they drink milk. But does the development of allergy depend on the amount of fat in the milk? In that case, we need to ask if the milk was whole milk, skim milk, 1%, or 2% fat? And does the person only drink one of those types of milk or multiple types of milk? Perhaps a person drinks 2% milk, but uses half-and-half in their coffee. So, we would need questions to understand that. In case people change their milk preferences over time, we might need questions to determine at what ages the person drank whole milk, for example, and then when did they start also drinking skim milk.

- But what if the issue of allergy is related to protein in milk? Then we need to be able to assess any other beverages and foods that contain milk protein. We cannot simply ask about milk. The range of foods with milk protein is tremendous and includes yogurt, milkshakes, and breads. Among breads alone, milk protein can be found in loaves of bread, biscuits, donuts, crackers, pancakes, waffles, French toast and others. Milk protein can be found in other foods, too, such as cereals and desserts, including cake, cookies, pudding, ice cream and pastries. Milk protein may be in scrambled eggs, butter, cream and margarine, salad dressing and even some "non-dairy" creamers. The list of foods with milk proteins goes on and on, even including meat products such as sausage, vegetables prepared *au gratin* or with butter or cream, candy including chocolate and many soups, chowders and bisques.

- It should be apparent that our simple question about milk is far more complicated than whether or not one drinks milk.

- Once we have identified all of the foods and beverages we need to ask about, we still need to determine the amount, or "dose," of milk consumed. This step can be very difficult. If you ask about eating soup that may have milk in it, how do you quantify if? A cup or a bowl? How big is the cup? Is the cup full to the top or about 2/3 of the way up? How much milk is in a "glass" of milk? We might need some tools to use, such as food models or empty containers, to show the person telling us the amount they consumed.

- And then if we agree on a way to standardize how large is a portion of soup or milk, how do we know that people are accurately reporting when they say they typically drink 3 glasses of milk per week? The answer is: if we want to come close to knowing the truth, then we have to demonstrate the validity of the questionnaire.

The validation process is separate from the research study and typically enrolls other people for the sole purpose of determining whether and how well the questionnaire works. One method is to ask people to fill out very detailed food diaries for a few different days (in nearly real-time as they are eating and drinking, so the information is fresh) and then compare how those same people answer a question a week later about what they consumed over the past week. The extent to which the answers using the two methods are in agreement provides evidence for the validity of the survey questions. Other approaches include asking people to take pictures of what they eat to use for validation. The main point is that there is a formal process of determining the validity of survey questions that is necessary if one wants to collect high quality data and be able to approximate the truth.

Certain of plaintiffs' experts have raised related issues in their critiques of the evidence for and against dose-response.[157] However, these same issues of validity of the exposure measure are just as important for assessing the overall proposition of whether or not talcum powder causes ovarian cancer. For example, Dr. Smith-Bindman criticizes Gates and other cohort studies for examining any talc use (which she labels "a weak, crude predictor").[158] But if Dr. Smith-Bindman believes the cohort studies suffer from assessing "any" use, she should apply this criticism even-handedly to the case-control studies and meta-analyses (such as the Penninkilampi study) that did the same. And she further should have pointed out that the questionnaires that case-control studies use to assess talc use habits are often haphazardly designed and not validated. Indeed, as Dr. Smith-Bindman observes, the Terry study (which numerous plaintiffs' experts rely on heavily) reported that the prevalence of powder use by controls in the underlying studies ranged from 15 to 45 percent, which she attributes to "variation in the definition of powder use" in the underlying studies it examined.[159] Her point affirms concern about the validity of talc exposure assessment and that the magnitude of error could be tremendous. But cohort studies are not uniquely subject to exposure assessment problems, and it is inappropriate for plaintiffs' experts to criticize them for this reason while ignoring similar issues with case-control studies.[160]

Further highlighting the importance of using validated measures of exposure, Dr. Colditz described the evolution of the Nurses' Health Study and noted that "there have been continuing efforts to validate questionnaire-based exposure measures used in the study."[161] For example, in order to measure nutritional exposures that might be relevant to cancer and other disease risks, Dr. Colditz noted that "[a]ssessment of long term diet is necessary to relate nutrient intake to the risk of chronic diseases," and that "this is best accomplished through the use of a food-frequency questionnaire." Further, he stated that the "Nurses' Health Study investigators have devoted great attention to the development, evaluation and refinement of food-frequency questionnaires

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157 Singh Rep. at 55.

158 Smith-Bindman Rep. at 21.

159 Smith-Bindman Rep. at 28.

160 *E.g.*, Moorman Dep. 187:13-18 (criticizing Gonzales 2016).

161 Colditz GA, Hankinson SE. The Nurses' Health Study: lifestyle and health among women. Nat Rev Cancer. 2005; 5(5):388-396. ("Colditz 2005").

for epidemiological applications." There were no such efforts employed in the NHS, nor in any other study, to develop and validate measures of talcum powder use.

Other authors have repeatedly discussed the limits of exposure measures in the epidemiologic studies. For example, in Schildkraut, the authors stated: "A recent publication of data from the WHI, which did not find an association with genital talc use and ovarian cancer, was accompanied by an editorial that emphasized the challenges in assessing the exposure to talc due to reliance on self-report. This limitation in the measurement of the exposure variables in the current study needs to be considered when interpreting our results."[162] And the Berge authors noted as a limitation to their meta-analysis that "neither the definition of the exposure of interest (genital talc use) nor the strategy for adjustment for potential confounders were fully consistent across studies."[163] Another limitation was the "self-reported information on the main exposure of interest, with no external validation."[164] In the Langseth (2008) paper, the authors noted that "the current body of epidemiologic evidence is insufficient to establish a causal association between perineal use of talc and ovarian cancer risk," and pointed to the "crudeness of the exposure metric used," and that "it is important that future studies, irrespective of study design, devote some effort to better assessment of exposure."[165] This "crudeness" of the exposure measure was apparent in Terry (2013) as the authors needed to define genital powder use as "any type of powder (talc, baby, deodorizing, cornstarch or unspecified/unknown)" and acknowledged that a study limitation was "differences in the wording of questions about genital powder use between studies."[166] In the same vein, another author cautioned that composition of body powders varies from one brand to the next. Thus, "[d]ata from additional cohort studies would be welcome, but without details concerning the composition of the powders used by cohort members—details that many participants may not be able to provide—the results of such studies may be similarly ambiguous in their interpretation."[167] Dr. Cramer, a plaintiffs' expert in prior talc cases, similarly acknowledged that "[t]here are inherent limitations quantifying a dose-response due to a lack of metrics for how much talc is in an 'application,' how much enters the vagina, and how much reaches the upper genital tract where, presumably, any deleterious effect is mediated."[168] Many other authors expressed similar concerns pertaining to the accuracy of exposure measurements.[169]

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[162] Schildkraut 2016 at 1416.

[163] Berge 2018 at 255.

[164] *Id.*

[165] Langseth 2008.

[166] Terry 2013 at 812, 820.

[167] Rosenblatt 2011.

[168] Cramer 2016 at 344.

[169] Gonzalez 2016 ("one challenge with studying talc is that the chemical formulation of talc has changed over time, and not all powders contain the mineral talc"); Cook 1997 ("it is not clear how ascertainment of perineal powder application correctly estimates actual exposure to particles in powder that may influence ovarian cancer risk"); Mills 2004 at 463 ("the lack of dose response between talc use and EOC may be explained by the inability to quantify the actual amount of talc used per application and timing of the application"); Rosenblatt 2011 ("the validity of all these studies, including ours, may be influenced by the level of non-response among cases and

In sum, without a validated measure of talcum powder use, it is impossible to correctly determine whether or not an exposure occurred or the quantity of purported exposure, making it impossible to reliably conclude that there is a causative relationship between perineal talcum powder use and ovarian cancer based on the current literature.

E. The Epidemiological Data Do Not Demonstrate Temporality.

The strongest evidence for temporality comes from studies that assess the exposure at one point in time, and then assess the outcome at a future time. The prospective cohort studies are the only studies in this matter that do that and thus represent the best evidence to assess temporality. As described more fully above, the cohort studies failed to show an association of ovarian cancer with talcum powder use. The case-control studies ask about past exposure, but they ask those questions at the same time that the outcome is already known. Temporality is assumed in case-control studies, though it is not a fact, as it is in cohort studies (or clinical trials). That is the reason that recall errors and recall bias are such a concern in case-control studies. Unlike prospective studies, subjects need to accurately remember and report past exposures. Recall bias occurs when people with a disease, compared to those without a disease, report different exposure histories compared to the truth. People with a disease may be more likely to recall or report exposures than those without the disease, which can inflate the apparent risk. This distortion is especially important when measured risks are low.

While it is a different concept from temporality, latency is a concept that is important to consider when evaluating temporality. Latency is the time from exposure to development of disease. When latency is known, one would want to make sure that not only did the exposure occur in the past, but that it occurred long enough ago in the past that a cancer would have time to develop. Obviously, without determining whether or not talcum powder causes ovarian cancer, it is not possible to state that there is a known latency. Nonetheless, Dr. Wolf states that the average latency period between exposure to talc and diagnosis of ovarian cancer is at least 20 years, citing two articles[170,171] that do not examine this issue.[172] Based on this theory, several experts have stated that a limitation of the cohort studies is that they were not of sufficient length to capture latency.[173] Obviously, without a known latency period, that concept is only speculative. Moreover, as explained above, the cohort studies have accounted for decades of talcum powder use. Thus, if women started using talcum powder at approximately 20 years

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controls, and by the potential for misclassification (differential and non-differential) of exposure status. The latter derives not just from errors in the recall of the use of genital powder, but from the fact that the presence or concentration of talc can vary from brand to brand and even within one brand of powder over time. Therefore, even when respondents are asked specifically about perineal exposure to powders that contain talc (as in our study), they may be unable to provide accurate information.").

[170] Purdie DM, Bain CJ, Siskind V, et al. Ovulation and risk of epithelial ovarian cancer. Int. J. Cancer. 2003; 104:228-232.

[171] Okada F. Beyond foreign-body-induced carcinogenesis: Impact of reactive oxygen species derived from inflammatory cells in tumorigenic conversation and tumor progression. Int. J. Cancer. 2007; 121:2364-2372.

[172] Wolf Rep. at 15.

[173] Singh Rep. at 11, 53; McTiernan Rep. at 47.

old[174] and the latency period is approximately 20 years,[175] then both the Women's Health Initiative Study and Sister Study would account for a sufficient latency period.

F. The Epidemiological Data Lack Coherence.

Dr. Hill stated that the cause and effect interpretation of the data "should not seriously conflict with the generally known facts and the biology of the disease." Dr. Hill cited the example of temporal trends in the rise in lung cancer rates while smoking was increasing. But here, there are no published studies that have demonstrated any such ecological coherence with talcum powder and ovarian cancer. Specifically, I can find no published studies that have examined trends in ovarian cancer rates in relation to trends in talcum powder use. Dr. Hill also cited, as an example of coherence, the changes of bronchial epithelial cells in smokers. But again, here there are no studies that have demonstrated histopathological differences in ovaries of talc users and non-users (nor in any tissues of the female genital tract). This fact strongly argues against coherence.

G. No Experimental Evidence.

There is no experimental evidence of the relationship of talcum powder use and ovarian cancer in humans, as plaintiffs' experts agree.[176]

H. The Epidemiologic Data Is Not Analogous.

A few of the experts considered analogy, although Dr. Moorman "did not weight it heavily"[177] and Dr. Singh found it "less significant than other viewpoints."[178] They and other experts opined that talcum powder's similarity to asbestos offers an appropriate analogy,[179] but asbestos and talc are distinct minerals, with distinct elemental composition and morphology, and it cannot simply be assumed that epidemiological study of asbestos can be applied by analogy to the case of talc, especially in light of the fact that talc itself has been extensively studied and its epidemiological literature reports vastly different risk levels than the asbestos literature. In particular, the talc/asbestos analogy is unpersuasive because talc exposure is not associated with an increased risk of mesothelioma or lung cancer (diseases caused by asbestos), and as set forth below, it is far from clear that asbestos causes ovarian cancer. Moreover, the limited analogy arguments that plaintiffs' experts advance do not make sense. For example, Dr. Moorman compares asbestos to "asbestiform talc," but fails to explain why her attempted analogy applies to platy talc.[180] Dr. Smith-Bindman similarly refers to talc's "fibrous nature," even though platy

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174 Cramer 2016 at 335.

175 Wolf Rep. at 15.

176 Moorman Rep. at 38; Singh Rep. at 66; Wolf Rep. at 16.

177 Moorman Rep. at 38.

178 Singh Rep. at 66.

179 Moorman Rep. at 38; Singh Rep. at 66; Smith-Bindman Rep. at 41.

180 Moorman Rep. at 38.

talc is not fibrous, and she further essentially concedes that the talc-ovarian cancer evidence is "weak[]" in making the unsupported claim that "weaker evidence" should suffice to prove causation when there is an appropriate analogy.[181] In short, analogy has not been established.

I. The Evidence For A Biological Mechanism By Which Talc Could Cause Cancer Is Weak.

Plaintiffs' experts generally propose that talc or alleged other constituents in talcum powder (e.g., asbestos, heavy metals or fragrance chemicals) can travel from the perineum up the genital tract to the ovaries – against gravity and the downward flow of vaginal mucous and menstrual fluids.[182] They also suggest an alternative pathway, via inhalation and the lymphatic system.[183] These proposed mechanisms are speculative and unsupported by science.

1. Studies Have Repeatedly Stated That Scientific Evidence Is Insufficient To Show Mechanisms Of Talc-Based Ovarian Carcinogenesis.

As an initial matter, based on my review of the available epidemiologic literature, many authors of studies have made clear that the evidence is insufficient to understand any purported mechanism by which talc-based cosmetic powders could cause ovarian cancer. For example:

Penninkilampi (2018)[184]

- "[T]he potential mechanism by which genital talc is associated with an increased risk of ovarian cancer hence remains unclear."
- "[U]nfortunately, the evidence remains insufficient to understand the mechanisms with any reasonable certainty."
- "[T]here is a substantial need for further research on a potential mechanism."

Berge (2018)[185]

- "[T]he biological basis and plausibility of a possible carcinogenic effect of talc on the ovaries is still not understood and remains questionable."

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181 Smith-Bindman Rep. at 41.

182 Carson Rep. at 8; Kane Rep. at 4, 14; McTiernan Rep. at 8, 58-59, 66; Moorman Rep. at 32-33; Plunkett Rep. at 27-38; Singh Rep. at 18-19, 57; Singh Dep. 212:6-18; Smith-Bindman Rep. at 35; Zelikoff Rep. at 12-14.

183 Carson Rep. at 8; Kane Rep. at 14; McTiernan Rep. at 58-59, 66; Moorman Rep. at 33; Plunkett Rep. at 27-28; Singh Rep. at 18-19, 57-58; Wolf Rep. at 11, 15; Zelikoff Rep. at 14-17.

184 Penninkilampi 2018 at 11-12, 14.

185 Berge 2018 at 255.

Cramer (2016)[186]

- "[U]nfortunately, no epidemiologic study of epithelial ovarian cancer and talc has taken the opportunity to determine whether talc can actually be found in tissues removed at surgery and correlated with exposure to talc."

Terry (2013)[187]

- "[T]he biological plausibility for the observed association between genital powder use and ovarian cancer has been challenged because evidence for dose-response has been inconsistent."
- "[L]ittle is known about the biologic effects of genital powder use."
- "[M]ore work is needed to understand how genital powders may exert a carcinogenic effect, and which constituents (e.g., talc) may be involved."

Gates (2008)[188]

- "The association remains controversial due to the lack of a clear dose-response with increasing frequency or duration of talc use, the possibility of confounding or other biases, and the uncertain biological mechanism."

Merritt (2008)[189]

- "[T]hese results in combination with previous studies suggest that chronic inflammation is unlikely to play a major role in the development of ovarian cancer."

Mills (2004)[190]

- "[R]esearch has provided little biologic or experimental evidence to support a relationship between talcum powder use and ovarian cancer risk."

Whittemore (1988)[191]

- "While these findings indicate that vaginal exposure to particulates can lead to deposition on the ovaries, they do not implicate such exposure in ovarian carcinogenesis, and data relating directly to this possibility are needed."

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186 Cramer 2016 at 344.

187 Terry 2013 at 819-20.

188 Gates 2008 at 2437.

189 Merritt 2008 at 174.

190 Mills 2004 at 464.

191 Whittemore 1988.

As these excerpts make clear, plaintiffs' experts' suggestion that biological plausibility is "accepted widely" based on "robust data" is simply false.[192]

2. Scientific Study Does Not Support The Inhalation Or Migration Theories By Which Talc Is Supposed To Reach The Ovaries.

Scientific data also fail to demonstrate a plausible mechanism by which talc or accessory particles could physically reach the ovaries from external use.

Plaintiffs' experts principally suggest that talc and asbestos particles can travel from the perineum up the genital tract to the ovaries – against gravity and the downward flow of vaginal mucous and menstrual fluids.[193] The results of research addressing retrograde transport have been inconclusive.[194] For example, one study examining the amount of talc in the ovaries of women who had undergone surgery for benign ovarian neoplasms found no correlation between the women's talc use and their talc particle counts.[195] Another study reviewed pathology slides from 213 ovarian tumors and found definite silicate crystals in only five patients, which may have reflected talc contamination from surgical gloves.[196] And as noted by IARC, while some studies of potential retrograde movement of particles in women who were about to undergo gynecological surgery for diseases or complications of the reproductive tract or organs have suggested that such transport is possible, "broad interpretations with regard to healthy women" based on these studies "may be limited."[197] Thus, IARC reported that, "[o]n balance, the Working Group believed that the evidence for retrograde transport of talc to the ovaries in normal women is weak."[198]

Relatedly, while plaintiffs' experts point out that talc particles and asbestos fibers have been found in ovarian tissue, this fact is of no scientific significance because researchers have found such particles in the ovaries of women with and without perineal talc use or other known exposures to talc or asbestos.[199] The Heller (1996) study found that "talc particles were observed to a similar extent with both exposed and unexposed subjects" and that particles were actually found in higher proportions among women who did not apply talc on the perineum, stating that "our results do not support a linear dose-related ovarian talc particle burden."[200] As this research

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192 Blair Smith Rep. at 20; Moorman Rep. at 32; Singh Rep. at 65.

193 Singh Dep. 212:6-18, 215:7 ("talc can migrate upwards").

194 IARC Talc Monographs at 392.

195 Heller 1996.

196 Yaker A, Benirschke K. A ten year study of ovarian tumors. Virchows Arch A Pathol Anat Histol. 1975; 366(4):275-86.

197 IARC Talc Monographs at 392.

198 *Id.* at 411.

199 Heller 1996 at 1508, 1510; Heller DS, Gordon RE, Westhoff C, Gerber S. Asbestos exposure and ovarian fiber burden. Am J Ind Med. 1996;2 9(5):435-439 (noting that asbestos fibers were found in ovarian tissue of women with and without history of exposure).

200 Heller 1996 at 1508, 1510.

indicates, the presence of fibers in ovarian tissue does not establish the relevant exposure pathways.

Studies have also failed to show an association between use of talc-dusted diaphragms and condoms and ovarian cancer.[201] Evaluating an association with the use of talc-dusted diaphragms and condoms has been deemed "the most valid method for testing the carcinogenic potential of talc" because "[b]y definition, the female reproductive tract is exposed to talc containing powders introduced by diaphragms, whereas an exposure route based on perineal dusting requires unproven assumptions about vaginal exposure."[202]

Moreover, numerous studies have considered whether tubal ligation and hysterectomy – procedures that "block the environmental contamination of the ovaries" – are associated with a decreased risk of ovarian cancer generally, while others have looked at this question in perineal talc users specifically.[203] Although plaintiffs' experts assert that these studies "strongly suggest that the increased risk of ovarian cancer associated with talcum powder products use is reduced or eliminated after tubal ligation or hysterectomy,"[204] my review of the available literature reveals that the results have been inconsistent.[205] In fact, the only cohort study to address this issue concluded that "no effect modification was seen by history of tubal ligation."[206] And a pooled analysis of case-control studies observed similar associations for talc use in women with tubal ligation or hysterectomy regardless of whether the "exposure to genital powder applications" occurred before or after the surgery.[207] Several case-control studies have found a lower incidence of ovarian cancer in patients who had tubal ligation but a higher incidence in patients who had hysterectomies,[208] which is a puzzling result since both hysterectomy and tubal ligation should cut off the pathway through which talc could travel to the ovaries. Because tubal ligation and hysterectomy would prevent the migration of talc particles from the perineum, the

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201 Hartge 1983; Fiume 2015 at 122S ("[S]tudies demonstrating that the use of talc-dusted condoms or diaphragms, which would clearly result in exposure close to the cervical opening, [have found that talc] was generally not associated with increased RR estimates for ovarian cancer."); Muscat & Huncharek 2008 at 5-6 (describing meta-analyses showing no association between use of talc-dusted diaphragms and condoms and ovarian cancer); Penninkilampi 2018 at 42, 44 ("Talc use on diaphragms or on sanitary napkins was not individually associated with increased risk of ovarian cancer.") .

202 Muscat & Huncharek 2008 at 5, 9 ("It may be argued that the overall null findings associated with talc-dusted diaphragms and condom use is more convincing evidence for a lack of a carcinogenic effect, especially given the lack of an established correlation between perineal dusting frequency and ovarian tissue talc concentrations and the lack of a consistent dose-response relationship with ovarian cancer risk.").

203 *Id.* at 7.

204 Smith-Bindman Rep. at 35.

205 Muscat & Huncharek 2008 at 7; Singh Rep. at 23 (admitting that the Terry pooled study found that "[a]fter excluding those with tubal ligation and hysterectomy, the results were similar. Restricting analysis to application before tubal ligation made no substantive difference.").

206 Gertig 2000 at 251.

207 Terry 2013 at 817.

208 Mills 2004 (finding odds ratios of 0.88 and 1.54 for tubal ligation/no tubal ligation and odds ratios of 1.79 and 1.33 for hysterectomy/no hysterectomy); Cramer 1999 at 352 (odds ratios of 0.98 and 1.80 for tubal ligation/no tubal ligation and 2.61 and 1.60 for hysterectomy/no hysterectomy).

fact that studies have not consistently shown a reduced risk associated with these surgeries undermines the premise that talc particles travel to the ovaries and cause cancer.

Finally, some of plaintiffs' experts espouse a theory that talc or accessory particles can reach the ovaries via inhalation (i.e., that women who use cosmetic talc inhale some amount of talc particles while they are applying cosmetic talc).[209] But I have not seen a mechanistic study that demonstrates that inhaled talc particles can reach the ovaries, and plaintiffs' experts concede there is not sufficient evidence pertaining to inhalation of talcum powder.[210] Furthermore, while most of the epidemiologic studies did not examine non-perineal application of talcum powder, those that assessed application to other body parts found inconsistent results. For example, although Penninkilampi found a small elevation in risk with "any non-perineal" talc use [1.24(1.01-1.51), this finding was limited by finding significant heterogeneity across the studies. In the Terry pooled analysis of more than 18,000 women, non-perineal application showed no risk [0.98(0.89-1.07)]. Likewise, the recent study by Cramer (2016) showed no association of body use of powder with ovarian cancer [0.99[0.84-1.16].

3. The Theory That Talc Can Cause Inflammation That Promotes Cancer Lacks Scientific Support.

The theory asserted by several of plaintiffs' experts that talc particles that reach the ovaries can cause inflammation leading to cancer (the "inflammation theory") also lacks support.[211]

First and foremost, no biological mechanism theory accounts for the fact that talc is not mutagenic or genotoxic.[212] This fact significantly undermines the theory that talc causes ovarian cancer, since gene mutation is widely recognized as what triggers ovarian cancer.[213] And Dr. Singh's assertion – without citation – that "[t]alc has also been shown to be mutagenic"[214] is simply incorrect, as is Dr. McTiernan's similar assertion that talc can cause genotoxicity.[215] In

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209 McTiernan Rep. at 66; Siemiatycki Rep. at 65; Moorman Rep. at 33; Singh Rep. at 19, 57-58.

210 Moorman Dep. 303:17-304:15 (stating there is not sufficient evidence to conclude that inhaled talcum powder causes ovarian cancer because there are not "epidemiologic studies that have actually looked at inhaled talcum powder in relation to ovarian cancer"); Singh Dep. 216:14-19 (agreeing that "studies of talcum powder use failed to show a statistically significant association between nongenital use of talcum powder and ovarian cancer").

211 Smith-Bindman Rep. at 12; McTiernan Rep. at 8; Siemiatycki Rep. at 65; Moorman Rep. at 33-34; Singh Rep. at 19.

212 Muscat & Huncharek 2008 at 9 (citing Endo-Capron S, Renier A, Janson X, et al. In vitro response of rat pleural mesothelial cells to talc samples in genotoxicity assays (sister chromatid exchanges and DNA repair). Toxicol In Vitro. 1993; 7(1):7-14); IARC Talc Monographs at 399.

213 Mayo Clinic, Cancer (Dec. 12, 2018), https://www.mayoclinic.org/diseases-conditions/cancer/symptoms-causes/syc-20370588; Anand P, Kunnumakkara AB, Sundaram C, et al. Cancer is a preventable disease that requires major lifestyle changes. Pharm Res. 2008; 25(9):2097-2116, 2098 (noting that "all cancers are a result of multiple mutations").

214 Singh Rep. at 19.

215 McTiernan Rep. at 67.

this vein, animal studies (including studies directly injecting talc into the ovaries of rats) have not shown that prolonged exposure to talc causes ovarian cancer or precancerous changes in ovarian cells.[216] Likewise, in vitro and pathological studies have not shown evidence of talc-induced ovarian cancer.[217]

The inflammation theory is also unsupported and implausible. A recent study sought to determine whether histological signs of inflammation were associated with ovarian cancer and found "no significant correlation . . . between serous carcinoma and histological signs of inflammation or chronic tubal injury."[218] Studies have not established a causal association between the use of cosmetic talc and cancers in vaginal, uterine and cervical tissue.[219] If talc (or alleged asbestos in talc products) produced inflammatory responses or carcinogenesis in ovarian tissue, it might also produce the same in other tissue. These tissues are closer to the perineum than the ovaries and likely are exposed to greater concentrations of talc than the ovaries.

The lack of evidence showing a reduced risk associated with the use of anti-inflammatory drugs further undermines the inflammation theory. Most meta-analyses examining this issue have found no risk reduction with either aspirin or NSAID use.[220] One did report a modest risk reduction for aspirin use but found no such reduction for non-steroidal anti-inflammatory drug ("NSAID") use.[221] The meta-analysis concluded that "[f]urther biological and pharmacological

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[216] Muscat & Huncharek 2008 at 9 (lifetime whole body exposure experiments in female laboratory rats found that ovarian tissue was not contaminated with talc and that ovarian tumor incidence was not increased) (citing Boorman GA, Seely JC. The lack of an ovarian effect of lifetime talc exposure in F344/N rats and B6C3F1 mice. Regul Toxicol Pharmacol. 1995; 21(2):242-243); Hamilton TC, Fox H, Buckley CH, et al. Effects of talc on the rat ovary. Br J Exp Pathol. 1984; 65(1):101-106 (study exposing rat ovaries to talc finding that the "epithelium covering the papillae was regular with no evidence of cytoplasmic or nuclear atypia"; there was no "evidence of frank neoplasia"; and that observed inflammation was not near the papillae).

[217] Muscat & Huncharek 2008 at 9; IARC Talc Monographs at 397-98; Lee P, Sun L, Lim CK, et al. Selective apoptosis of lung cancer cells with talc. Eur Respir J. 2010; 35(2):450-452, 452; Nasreen N, Mohammed KA, Brown S, et al. Talc mediates angiostasis in malignant pleural effusions via endostatin induction. Eur Respir J. 2007; 29(4):761-769, 761-762 (in vitro studies reporting that talc stops new blood vessels from forming and causes cell death only in malignant cells, leaving healthy cells alone).

[218] Malmberg K, Klynning C, Flöter-Rådestad A, Carlson JW. Serous tubal intraepithelial carcinoma, chronic fallopian tube injury, and serous carcinoma development. Virchows Arch. 2016; 468(6):707-713.

[219] Singh Dep. 209:9-16.

[220] Bonovas S, Filioussi K, Sitaras NM. Do nonsteroidal anti-inflammatory drugs affect the risk of developing ovarian cancer? A meta-analysis. Br J Clin Pharmacol. 2005; 60(2):194-203 (RR 0.93 (95% CI: 0.81-1.06) for aspirin use; RR 0.88 (95% CI: 0.76-1.01) for NSAID use); Ni X, Ma J, Zhao Y, et al. Meta-analysis on the association between non-steroidal anti-inflammatory drug use and ovarian cancer. Br J Clin Pharmacol. 2013; 75(1):26-35 (RR 0.94 (95% CI: 0.87-1.01) for aspirin use; RR 0.89 (95% CI: 0.74-1.08) for NSAID use); Baandrup L, Faber MT, Christensen J, et al. Nonsteroidal anti-inflammatory drugs and risk of ovarian cancer: systematic review and meta-analysis of observational studies. Acta Obstet Gynecol Scand. 2013; 92(3):245-255 (RR 0.93 (95% CI: 0.84-1.02) for aspirin use; RR 0.94 (95% CI: 0.84-1.06) for NSAID use).

[221] Trabert B, Ness RB, Lo-Ciganic WH, et al. Aspirin, nonaspirin nonsteroidal anti-inflammatory drug, and acetaminophen use and risk of invasive epithelial ovarian cancer: a pooled analysis in the Ovarian Cancer Association Consortium. J Natl Cancer Inst. 2014; 106(2):djt431, 5 (2014) (for aspirin, OR 0.91 (95% CI: 0.84-0.99); for NSAIDs, OR 0.90 (95% CI: 0.77-1.05)).

research is necessary to understand the mechanisms of ovarian cancer risk reduction by aspirin use."[222] The authors reported the results of further study just this year, continuing to find a modest decrease in risk with daily aspirin use but not with other types of anti-inflammatories, and further contradicting the inflammation theory, "observ[ing] a consistently elevated ovarian cancer risk with frequent, long-duration use of aspirin and nonaspirin NSAIDs.[223] Moreover, the Wu 2009 study – on which plaintiffs' experts have relied on the issue of dose-response – likewise found the opposite effect, reporting that, "contrary to the study hypothesis that NSAIDs may have chemopreventative effects by decreasing inflammation, we found that the risk of ovarian cancer ***increased*** significantly with increasing frequency and duration of NSAIDs use."[224] And Merritt (2008) additionally found risk reduction with the use of anti-inflammatories, concluding that "on balance, chronic inflammation does not play a major role in the development of ovarian cancer."[225] In sum, and as plaintiffs' experts agree, studies of the effect of anti-inflammatory drugs on ovarian cancer are mixed at best, and some even show the reverse relationship – i.e., increased incidence of ovarian cancer with increased use of NSAIDs.[226]

Finally, "inflammation" is a broad term and does not inevitably lead to cancer. For example, pollen can lead to increased inflammation in the asthmatic lung, but it does not cause cancer. Thus, even if one finds inflammation in tissue, that does not mean that cancer inevitably or even likely follows from that. And if talc in fact caused cancer by causing inflammation, it would surely do so in patients who undergo pleurodesis (which entails the therapeutic injection of talc into the pleural cavity to cause beneficial scarring). Yet, there is no evidence that pleurodesis patients subsequently develop cancer as a result of the procedure. Plaintiffs' expert Dr. Ghassan Saed has performed experiments – apparently for litigation purposes[227] – to attempt to establish an inflammation-based mechanism by which talc could cause ovarian cancer. While I leave a detailed assessment of Dr. Saed's efforts to other experts, I did review Dr. Saed's report and his two depositions and was struck by the irregularities in his study, which render his results highly questionable. I also read the highly skeptical comments from the reviewers at *Gynecologic Oncology*, which rejected his manuscript.[228] But even accepting the results of Dr. Saed's study, they at best raise questions about the inflammation hypothesis that would have to be addressed through future *in vitro* and *in vivo* testing, as he effectively acknowledged at his

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222 *Id.*

223 Trabert B, Poole EM, White E, et al. Analgesic Use and Ovarian Cancer Risk: An Analysis in the Ovarian Cancer Cohort Consortium, J. Nat'l Cancer Inst. 2019; 111(2):137-145, 139-142 (emphasis added).

224 Wu 2009 (emphasis added).

225 Merritt 2008.

226 Singh Dep. 231:23-233:2 ("[NSAIDs] don't consistently reduce the risk of ovarian cancer"); Kane Rep. at 9-13 ("[S]ome studies show[] a protective effect of anti-inflammatory drugs on the risk of developing carcinoma, although some studies have failed to show a protective effect."); Blair Smith Rep. at 17-18 (describing studies that "looked at the effects of aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) on the risk of developing cancer" as "inconsistent").

227 Saed Dep. Vol. I 62:16-63:7, 72:10-73:2, 178:14-21.

228 Gynecologic Oncology Email dated Sept. 19, 2018 re: GYN-18-1020: Final Decision.

deposition.[229]

VII. THE ASBESTOS LITERATURE DOES NOT SUPPORT THE THEORY THAT ASBESTOS ALLEGED TO BE IN COSMETIC TALC COULD CAUSE OVARIAN CANCER.

There are numerous problems with plaintiffs' experts' theory that asbestos is an accessory mineral present in cosmetic talc that causes ovarian cancer.

First, all of the problems addressed above with respect to plaintiffs' theories by which particulates in talcum powder could migrate to the ovaries would apply to asbestos fibers. And plaintiffs' experts' inhalation theories are all the more infirm with respect to asbestos particularly. Assuming talc contained asbestos, the larger burden of any inhaled asbestos should be seen in the lungs, which are directly exposed, rather than the ovaries, which would only be indirectly exposed, if at all. If that is the case, as Dr. Moorman testified, we should be seeing an epidemic of mesothelioma and lung cancer cases among cosmetic talc users.[230] But no expert has identified any studies showing that mesothelioma or lung cancer is a risk of talc use, and I am not aware of any such studies. To the contrary, studies that have looked at talc miners and millers – who would presumably confront greater exposures to asbestos if it were present in talc given the occupational context – have not found any increased incidence of mesothelioma or lung cancer attributable to talc exposure in the mines or mills.[231] Notably, IARC emphasized this point, stating that there was "little or inconsistent evidence of an increased risk of cancer in the studies of workers occupationally exposed to talc," where the potential for talc inhalation would be particularly significant, and that "studies of talc miners and millers were considered to provide the best source of evidence."[232] And the body of literature investigating perineal talc use has focused on ovarian cancer, and not mesothelioma or lung cancer, which indicates that researchers have not even considered them worth investigating.

In addition to the lack of a plausible mechanism by which asbestos could reach the ovaries, there is also a lack of any reliable epidemiology supporting such a causal connection. There have been relatively few studies examining the association between asbestos exposure and

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229 Saed Dep. Vol. II 542:16-25.

230 Moorman Dep. 112:7-15 ("Q. You would agree with me that if talcum powder, that is used in cosmetic talc products, is, in fact, contaminated with asbestos, then you would expect to see increased cancer incidence rates, for example, of mesothelioma, in cosmetic talc miners and millers; correct? . . . . [A] I wouldn't be surprised to see that, yes.").

231 Fiume 2015 at 119S (studies looking at occupational inhalational talc exposure do not show an increased risk of lung disease); Pira PE, Coggiola M, Ciocan C, et al. Mortality of Talc Miners and Millers from Val Chisone, Northern Italy: An Updated Cohort Study. J Occup Environ Med. 2017; 59(7):659-664 (concluding that there was a lack of association between exposure to asbestos-free talc, lung cancer, and mesothelioma in a cohort of talc miners and millers from Val Chisone, Italy); Wergeland E, Andersen A, Baerheim A. Morbidity and mortality in talc-exposed workers. Am J Ind Med. 1990; 17(4):505-513 (finding no elevated incidence of lung cancer or mesothelioma in a cohort or 94 talc miners and 295 talc millers).

232 IARC Talc Monographs at 412.

ovarian cancer.[233] Of the studies that have reported a statistically significant association between asbestos exposure and ovarian cancer, all looked at populations heavily exposed to asbestos in the workplace.[234] As noted by the authors of a 2011 meta-analysis that included most of this research, studies examining the asbestos-ovarian cancer association have been "limited," in part due to a "[s]mall number of cases" – i.e., "[m]uch fewer women than men have been exposed to asbestos, particularly in [the] more heavily exposed occupational settings" that have predominantly been examined.[235] Although some of these studies show a statistically significant elevated risk, others do not, and the overall results are highly inconsistent.[236] Moreover, the meta-analysis calculated an overall standardized mortality ratio ("SMR") of 1.75 across 16 studies, which is not even a doubling of risk.[237] The SMR in these studies ranged from 0.79 in a study of Polish women diagnosed with asbestosis (in which there was only one case of ovarian cancer across 490 exposed women) to 4.77 in a study of Italian women compensated for asbestosis (nine cases of ovarian cancer in 631 exposed women).[238] Ten of the 16 studies reported SMRs lower than 2.0, none of them statistically significant.[239]

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233 International Agency for Research on Cancer, Monographs on the Evaluation of Carcinogenic Risks to Humans Vol. 100C: Asbestos (Chrysotile, Amosite, Crocidolite, Tremolite, Actinolite, and Anthophyllite) 253 (2012) ("IARC Asbestos Monographs") (observing that "the published literature examining the association between asbestos exposure and cancer of the ovaries is relatively sparse").

234 Acheson ED, Gardner MJ, Pippard EC, Grime LP. Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow-up. Br J Ind Med. 1982; 39(4):344-348 (for gas mask workers exposed to crocidolite, SMR 2.75 (95% CI: 1.42-4.81)); Berry G, Newhouse ML, Wagner JC. Mortality from all cancers of asbestos factory workers in east London 1933-80. Occup Environ Med. 2000; 57(11):782-785 (for insulation workers, SMR 2.53 (95% CI: 1.16-4.80)); Camargo MC, Stayner LT, Straif K. Occupational exposure to asbestos and ovarian cancer: a meta-analysis. Environ Health Perspect. 2011; 119(9):1211-1217, 1216 ("Camargo 2011") (meta-analysis "restricted to highly exposed women" reporting "findings . . . consistent with the hypothesis that exposure to asbestos is associated with an increased risk of ovarian cancer"); Germani D, Belli S, Bruno C, et al. Cohort mortality study of women compensated for asbestosis in Italy. Am J Ind Med. 1999; 36(1):129-134 ("Germani 1999") (for cement works, SMR 5.40 (95% CI: 1.75-12.61); for textile works, SMR 5.26 (95% CI: 1.43-13.47); for all workers, SMR 4.77 (95% CI: 2.18-9.06)); IARC Asbestos Monographs at 256 (concluding that there is a causal association based "on five strongly positive cohort mortality studies of women with ***heavy occupational exposure*** to asbestos") (emphasis added); Magnani C, Ferrante D, Barone-Adesi F, et al. Cancer risk after cessation of asbestos exposure: a cohort study of Italian asbestos cement workers. Occup Environ Med. 2008; 65(3):164-170 (for cement factory workers, SMR 2.27 (95% CI: 1.04-4.32)); Wignall BK, Fox AJ. Mortality of female gas mask assemblers. Br J Ind Med. 1982; 39(1):34-38. ("Wignall & Fox 1982") (for gas mask workers, SMR 2.13).

235 Reid A, de Klerk N, Musk AW. Does exposure to asbestos cause ovarian cancer? A systematic literature review and meta-analysis. Cancer Epidemiol Biomarkers Prev. 2011; 20(7):1287-129, 1287 ("Reid 2011").

236 *See id.* ("The relationship between asbestos exposure and ovarian cancer is not as well understood."); *see also id.* at 1293 fig. 1 (chart showing the 16 studies, 12 of which did not report statistically significant results); *id.* at 1294 ("The present study has shown that 4 of 14 cohort studies reported a statistically significant excess rate for ovarian cancer among women exposed to asbestos. Of the remaining 10 studies, 5 reported a tendency to excess but failed to reach statistical significance and 5 reported rates that were similar to those of their reference populations. Strong evidence of consistency was not observed among these studies, although no study reported any protective effect."); IARC Asbestos Monographs at 254-56 (describing cohort studies and case-control studies).

237 Reid 2011 at 1287 (abstract).

238 *Id.* at 1289.

239 *Id.* at 1289-90.

Addressing this body of research, the authors of the 2011 meta-analysis noted above acknowledged an IARC Working Group's recent conclusion that a causal association between asbestos exposure and ovarian cancer had been established,[240] but criticized that conclusion as "premature and not wholly supported by the evidence."[241] The authors also emphasized that "[s]trong evidence of consistency was not observed among these studies,"[242] pointing out that "no significant excess risk was reported among those studies that examined the incidence of ovarian cancer where cases were ascertained from a cancer registry" as opposed to from death certificates, which is significant because there is evidence of misclassification in death certificates.[243] The authors also noted that many studies involved too few women to address dose-response.[244] With respect to the studies that did address dose-response, the findings "were inconsistent"; no study showed a "statistically significant trend of ovarian cancer with degree of asbestos exposure"; and "there was no evidence of a significant trend across studies as grouped exposure increased."[245] In light of these conclusions, I find it puzzling that some of plaintiffs' experts claim reliance on this meta-analysis for the conclusion that "[a]sbestos has been established as a cause of . . . epithelial ovarian cancer."[246] The study itself claims the opposite.

In addition, no study has found that asbestos exposure comparable to that allegedly sustained by women who use cosmetic talc causes an increased risk of ovarian cancer. Specifically, the occupational studies described above include workers who worked with raw asbestos as part of their job for months or years at a time.[247] And as Dr. Moorman testified, the level of exposure is qualitatively different in the occupational context from the exposure to the genital areas alleged by plaintiffs.[248] I am not aware of any study showing that the use of cosmetic talc would result in asbestos exposures comparable to occupational asbestos exposure even if the cosmetic talc contained trace amounts of asbestos, as claimed by plaintiffs' experts. Thus, the results of occupational studies cannot be reliably extrapolated to exposure scenarios such as cosmetic talc use.

The results of the occupational asbestos studies also cannot be used to support causation of ovarian cancer in cosmetic talc users because the studies have predominantly examined exposure to crocidolite asbestos or some combination of crocidolite and chrysotile, and

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240 IARC Asbestos Monographs at 256.

241 Reid 2011 at 1294.

242 *Id.*

243 *Id.* at 1293-94.

244 *Id.* at 1294.

245 *Id.*

246 McTiernan Rep. at 57; McTiernan Dep. 268:21-25; Moorman Dep. 108:6-109:10, 111:7-14.

247 Germani 1999 at 129 ("Subjects included in this cohort were certainly exposed to high levels of asbestos."); Wignall & Fox 1982 at 35 (subjects were "directly exposed to asbestos dust," and "by the end of the working day they were covered in fluff from the pads" they worked on).

248 Moorman Dep. 106:4-17.

crocidolite is regarded as the most potent form of asbestos.[249] I note that studies examining the composition of talc-based body powders have not observed crocidolite fibers.[250]

Even assuming exposure to asbestos of some variety and in certain exposure scenarios can cause ovarian cancer, no science supports the notion – put forth by a number of plaintiffs' experts – that "any exposure" to asbestos can cause ovarian cancer.[251] To the contrary, as suggested by the discussion of occupational studies above, the available data suggest that very significant exposure would be necessary. This conclusion is strongly supported by the fact that the few studies that have looked at environmental asbestos exposure (in women living in an asbestos mining town and family members of male asbestos factory workers) rather than occupational exposure do not show a statistically significant increased rate of ovarian cancer or increased mortality from ovarian cancer.[252] For example, in one study of women who lived near or worked in a crocidolite mine and who had cumulative exposures of up to 40 fiber/cc-years, there was no increased risk of ovarian cancer.[253] Even these studies are not perfectly analogous to the asbestos exposure alleged through perineal use of cosmetic talc. But they underscore the fact that not every circumstance where there is asbestos exposure, even crocidolite exposure, leads to elevated ovarian cancer risk.

Finally, I note that studies addressing whether there is an association between asbestos and ovarian cancer have cautioned that to the extent there is an observed association, it may be inflated by the misclassification of other diseases such as mesothelioma as ovarian cancer on subjects' death certificates.[254] As these studies have explained, it has only recently become

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249 Reid 2011 at 1291 (noting that crocidolite is "the most mesotheliogenic of the asbestos fibers"); IARC Asbestos Monographs at 242 (discussing studies finding no excess mortality for cancer of the pharynx in amosite asbestos miners but an excess mortality rate for crocidolite miners and a higher risk rate for factory workers exposed to crocidolite than workers exposed to chrysotile); *id.*at 254-55 (relying on studies that involved crocidolite and, in some cases also chrysotile).

250 IARC Talc Monographs at 303-05.

251 *E.g.*, Moorman Dep. 75:22-76:3.

252 Reid A, Heyworth J, de Klerk NH, Musk B. Cancer Incidence Among Women and Girls Environmentally and Occupationally Exposed to Blue Asbestos at Wittenoom, Western Australia. Int J Cancer. 2008; 122(10):2337-2344 (study of 2,552 women living in an asbestos mining town in Australia (reporting a "minimum estimate" standard incidence ratio ("SIR") of 1.11 (95% CI 0.39-1.84) and "maximum estimate" SIR of 1.43 (95% CI 0.50-2.37), depending on the method used to determine when to stop following women in the study; a standard incidence ratio reports the ratio of the number of cases of cancer found in the studied population relative to the expected number of such cases as derived from broader population statistics rather than a control group, and a standard mortality ratio ("SMR") employs a similar comparison but focuses on rates of death rather than incidence of disease); Reid A, Segal A, Heyworth JS, et al. Gynecologic and breast cancers in women after exposure to blue asbestos at Wittenoom. Cancer Epidemiol Biomarkers Prev. 2009; 18(1):140-147 ("Reid 2009") (analysis of ovarian cancer incidence in the same population (SIR 1.18 (95% CI: 0.45-1.91))); Ferrante D, Bertolotti M, Todesco A, et al. Cancer mortality and incidence of mesothelioma in a cohort of wives of asbestos workers in Casale Monferrato, Italy. Environ Health Perspect. 2007; 115(10):1401-1405 (study of family members of men employed at an asbestos-cement factory in Italy (SMR 1.42 (95% CI: 0.71-2.54))).

253 Reid 2009.

254 Reid 2011 at 1287 (explaining that many studies ascertained mortality from death certificates, "[t]he accuracy of [which] has been questioned repeatedly"; observing that it has been "particularly difficult to distinguish

technologically possible to reliably "distinguish pathologically between peritoneal mesothelioma and ovarian cancer."[255] As the authors of one meta-analysis explained, even a low number of misclassification errors can drastically affect reported mortality rates given the limited number of ovarian cancer cases in the studies.[256] Notably, the authors of that meta-analysis did not find a statistically significant ovarian cancer incidence when looking only at studies that obtained ovarian cancer diagnoses from cancer registries rather than death certificates.[257]

VIII. HEALTH CANADA AND THE ANALYSIS BY MOHAMED TAHER

I understand that plaintiffs' experts have begun relying on the recent draft screening assessment of talc by Health Canada[258] and the related analysis by Mohamed Taher[259] and others. I have reviewed these documents, and they are consistent with the opinions I set forth above and do not support a conclusion that talc causes ovarian cancer.

The Health Canada ("HC") assessment raises a number of new issues that if anything further cloud the scientific picture and erect further obstacles to a conclusion that perineal talcum powder use causes ovarian cancer. For example, the document highlights other sources of exposure to talc. Specifically, it states that "a potential concern for human health has been identified" for perineal exposure to talc "from use of various self-care products (e.g., body powder, baby powder, diaper and rash creams, genital antiperspirants and deodorants, body wipes, bath bombs)."[260] The document further notes that talc is present in approximately 8,500 self-care products, in addition to being found as a food additive, in medications, and many other consumer and commercial products.[261]

In other respects, the HC assessment largely covers old ground. Indeed, as part of the overall assessment, there was a health effects assessment that relied on the work of other

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between peritoneal mesothelioma and ovarian serous carcinoma"). Notably, this meta-analysis found that "no significant excess risk was reported among those studies that examined the incidence of ovarian cancer where cases [were] ascertained from a cancer registry." *Id.* at 1294.

255 Camargo 2011 at 1216; *see id.* at 1215 (observing that earlier meta-analyses concluded that they could not conclude causality despite evidence of an association because of concerns about tumor misclassification and failure to account for known risk factors).

256 Reid 2011 at 1294 ("Where disease outcome was identified from the cause of death as listed on the death certificate, given the small numbers of ovarian cancer cases in each study, even misclassification of 1 cancer may exert a large impact on the exposure effect.").

257 *Id.* ("The meta-analysis of those studies that examined ovarian cancer as determined on the death certificate reported an excess risk. In contrast, no significant excess risk was reported among those studies that examined the incidence of ovarian cancer where cases [were] ascertained from a cancer registry.").

258 *See* Health Canada, Draft screening assessment talc ($Mg_3H_2(SiO_3)_4$), Chemical Abstracts Service Registry Number 14807-96-6, https://www.canada.ca/content/dam/eccc/documents/pdf/pded/talc/Draft-screening-assessment-talc.pdf ("HC Assessment").

259 Taher 2018.

260 *See* HC Assessment at iii.

261 *See id.* at 6.

agencies (e.g., IARC and the United States Environmental Protection Agency) and a literature search. With regard to perineal exposure to talc, the HC assessment cites IARC's Group 2B classification (possibly carcinogenic to humans), and the CIR Expert Panel (2013) that "determined that there is no causative relationship between cosmetic use of talc in the perineal area and ovarian cancer[.]"[262] The assessment notes that rodents are poor experimental models for perineal studies and that "animal data are very limited."[263] In terms of human studies, it cites several meta-analyses, including those cited by plaintiffs' experts, as well a newer unpublished manuscript by Taher (2018).[264]

In a discussion of mode of action, the HC assessment states that "the etiology of most ovarian tumors, in general, has not been well established."[265] While it notes that talc particles instilled into the uterus or to a lesser extent the vagina can be found in the ovaries of rats, no similar translocation occurred in studies of rabbits and monkeys.[266]

The HC assessment found no health effects of ingested talc or dermally applied talc. With regard to inhalation, it cites the Danish EPA (2016) "note that talc is not absorbed via inhalation."[267] It points to potential for retention of talc in the lungs as leading to talc-induced pneumoconiosis or talcosis in certain industrial settings.[268] The assessment considers the NTP rat study of inhalation (1993) of talc with doses as high as 18 mg/m^3.[269] It cites conclusions of a symposium of experts from the NTP as well as academic, industry and government experts who evaluated the NTP study results and reached a consensus that because the dose was so high, the neoplasms seen were not relevant to human health risk assessment.[270] The lung tumors seen in only female rats were judged to be attributed to the general particle effects of dust, and not specific to talc, and the pheochromocytomas were attributed to tissue hypoxia, and not talc per se.[271]

The HC assessment also addresses the issue of asbestos, noting that selective mining, ore processing, and benefaction can remove many of the impurities from mined talc, that United States Pharmacopeia ("USP") requires the absence of asbestos, and that cosmetic grade talc should comply with USP standards.[272] Further, "health effect studies on cosmetic-grade talc

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262 *Id.* at 15.

263 *Id.*

264 *Id.* at 16-17.

265 *Id.* at 18.

266 *Id.* at 18-19.

267 *Id.* at 11.

268 *Id.*

269 *Id.* at 12.

270 *Id.* at 13.

271 *Id.*

272 *Id.* at 3.

cited in this assessment were considered to be free of asbestos."[273]

At the end of the day, the HC assessment failed to conclude that talc use causes ovarian cancer,[274] and plaintiffs' experts misread the report to the extent they contend that it did.[275]

HC's overall assessment appears to rely heavily on the unpublished meta-analysis by Taher and cites Taher's Bradford Hill analysis extensively. HC's extensive reliance on Taher is unusual and problematic. First, the manuscript has not gone through the peer-review process for publication. There is no way to know, at this point, whether and where it will ultimately be published, but even if it is, there is no assurance that the findings and conclusions will be the same once reviewers and editors have provided feedback. Second, it seems unusual to rely on the Taher paper in that there is no novelty and the studies reviewed in it have been repeatedly evaluated by other authors, whose results have, in fact, been through the peer-review process.

As a rationale for performing the study, Taher et al. cite "increasing concern that perineal exposure to talc, a commonly used personal care product, might be associated with an increased risk of ovarian cancer."[276] Further, they note that "the data describing this association is somewhat inconsistent."[277] Again, it is not clear why another meta-analysis of the same underlying data would be expected to solve past inconsistency.

In reviewing animal studies, Taher et al. note that "data from the animal studies [that] considered various routes of talc administration are inconsistent. . . ."[278] They cite the NTP rat study (1993), findings that HC stated "were not relevant to human health risk assessment" and were not specific to talc.[279] Taher et al. use the NTP study as evidence that, "overall, the avaiable [sic] experimental data suggest irritation, followed by oxidative stress and inflammation, may play be involved [sic] in local carcinogenic effects of talc in the ovaries."[280] Taher et al. note that "data on talc migration in the genital tract of animals is inconsistent, but could not exclude such possibility."[281]

The Hill analysis performed by Taher et al. also has serious flaws. With respect to strength of association, the authors note that 6 of 30 studies showed statistically significant risk

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273 *Id.*

274 *Id.* at 28 (concluding that talc use is a "potential concern for human health").

275 Moorman Dep. 145:19-21.

276 Taher 2018 at 2.

277 *Id.* at 3.

278 *Id.* at 22.

279 HC Assessment at 3.

280 Taher 2018 at 23.

281 *Id.* at 24.

of 1.5 or greater and that none of the cohort studies found statistically significant associations.[282] While these findings show marked inconsistency, they are not supportive of a strong association.

With regard to consistency, Taher et al. cite 15 of 30 studies with positive, significant associations.[283] Obviously, the same number do not show such an association, which is further evidence against consistency.

As to temporality, Taher et al. state that "the participants recalled that exposure to talc preceded the reported outcome,"[284] which ignores the fact that this recall is retrospective rather than prospective.

Regarding biologic gradient, the cited evidence is that 6 of 12 studies showed a significant dose-response trend,[285] which is again evidence of the inconsistency of the study findings, and is in any event wrong given that the cited positive studies included several that did not find a dose response with cumulative use, such as Mills 2004 and Rosenblatt 2011. Moreover, at a later point in the paper, the authors acknowledge that "conflicting findings were reported on the nature of the exposure-response relationship" and that a possible increasing trend is hampered by "a high degree of uncertainty surrounding many of the risk estimates."[286]

For experimentation, there are no cited human studies and no tests of animal models of perineal talc and ovarian cancer. The authors again cite the NTP rat study,[287] which remains problematic for the reasons discussed previously.

The analysis of analogy relies on supposed similarities of talc and asbestos and the belief that there are histologic similarities of ovarian cancer and mesothelioma, and that these purported similarities have some bearing on talc causing cancer (even though Taher et al. state that "talc is not genotoxic").[288]

In the discussion, the authors note subgroup differences they observed by ethnicity, menopausal state, and tubal ligation. But they go on to note that these three subgroup analyses (ethnicity, menopausal state and pelvic surgery) showed considerable heterogeneity that "might have had an impact on the results."[289]

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282 *Id.* at 25.

283 *Id.*

284 *Id.* at 26.

285 *Id.*

286 *Id.* at 37.

287 *Id.* at 26.

288 *Id.* at 27.

289 *Id.* at 30.

Taher et al. reaffirmed the effect of study design on results, with, once again, positive findings only in population-based case-control studies, but not in those with hospital-based controls [0.96 (0.78-1.17)] or in cohort studies [1.06 (0.9-1.25)]. They also highlighted previously demonstrated paradoxical findings, such as lower risk of cancer with longer use of talc and the "expected, yet non-significant, negative association" with talc applied to diaphragms.[290] While they noted a protective effect of tubal ligation [0.64 (0.45-0.92)], they acknowledged incoherent findings of no significant effect of hysterectomy [0.89 (0.54-1.46)] and a small, non-significant higher risk in women with both tubal ligation and hysterectomy [1.06 (0.78-1.42)].[291]

In the conclusion, the authors state that their evaluation is consistent with that of IARC in 2010 and that it "indicates that perineal exposure to talc powder is a possible cause of ovarian cancer in humans."[292] In other words, eight years later their conclusion is the same – that the evidence shows only that it is possible, not probable, that perineal talc use causes ovarian cancer. Thus, Taher does not add anything new to the body of literature addressed in this report.

IX. CONCLUSION

It is my opinion, based on my qualifications and my extensive review of the available epidemiology studies and scientific literature, that there is not sufficient evidence to conclude that there is a causal relationship between perineal talcum powder exposure and ovarian cancer. The epidemiologic literature shows a non-existent association or, at most, a small association between perineal talc use and ovarian cancer that constitutes only weak epidemiologic evidence that can be attributed to bias, confounding or chance. The studies are inconsistent across study designs and within study designs, as cohort and hospital-based case-control studies do not show a statistically significant association and only a subset of the population-based case-control studies demonstrate a statistically significant association. Moreover, the case-control studies do not show any consistent evidence of a dose-response relationship, and there is a complete lack of evidence for dose-response in the cohort studies. The theories pertaining to biological plausibility are entirely speculative and have not been demonstrated in the epidemiology studies or scientific literature; rather, relevant science contradicts the purported theories of talcum powder transport and development of ovarian cancer by inflammation. Finally, the assertion that asbestos present in talc – even if true – causes ovarian cancer is problematic on the grounds that there is a lack of a plausible mechanism by which asbestos could reach the ovaries and also a lack of any reliable epidemiology supporting such a causal connection.

All of the opinions in this report are stated to a reasonable degree of scientific certainty.

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290 *Id.* at 32.

291 *Id.* at 33.

292 *Id.* at 49.

APPENDIX A

Appendix A: Sample Of Pre-2014 News Articles Addressing Posited Link Between Talc Use And Ovarian Cancer

[1] Boston Globe, Study links talc use to ovarian cancer (Aug. 6, 1982)

[2] New York Times, Talcum company calls study on cancer link inconclusive (August 12, 1982) ("A major talcum powder manufacturer, while criticizing a recent study linking the use of talcum powder by women to ovarian cancer, said it would further investigate any possible relationship between cosmetic-grade talc and the development of disease.")

[3] New York Times, Personal Health, (July 03, 1985) ("A number of studies have indicated that exposure to talc, the principal ingredient in talcum powder, increases the risk. This may be because talc is usually contaminated with particles of asbestos, which are known cancer-promoting substances.")

[4] Washington Post, Fighting Ovarian Cancer: Doctors Don't Know Who's at Risk, or Why (May 30, 1989) ("One theory is that asbestos containing talc may account for some of the increased rate of disease in western countries that emphasize personal hygiene.")

[5] San Francisco Chronicle, Use powder with caution (July 31, 1990)

[6] Business Times, Safe Neways option to looking beautiful (Feb 17, 1991) ("According to him, the talc in talcum powder and colour cosmetics have a similar molecular structure as asbestos which can cause ovarian cancer while the alcohol content in mouthwash can cause throat and stomach cancer.")

[7] Philadelphia Inquirer, Cancer risk and talcum linked; for women who used talc for a lifetime, the risk increased 300% (July 1, 1992)

[8] Houston Chronicle, Use of talc on panties tied to cancer (July 1, 1992)

[9] Los Angeles Daily News, Study says talc use increases women's risk of ovarian cancer (July 1, 1992)

[10] Seattle Times, Study links talcum use, ovarian cancer (July 1, 1992)

[11] St. Louis Post-Dispatch, Talcum powder, ovarian cancer linked (July 2, 1992)

[12] The Independent, Condom talc risks, (Mar. 21, 1995) ("They point out that if it gets into the female reproductive tract, talc may result in fallopian tube fibrosis and infertility, and it may also be linked to ovarian cancer.")

[13] Philadelphia Inquirer, Breaking the silence: women take on a deadly stalker ovarian cancer will kill more than 14,000 this year. Activists are targeting ignorance and complacency (May 18, 1997) ("using talcum powder on the genital area, among other factors, increase the risk")

[14] Chicago Tribune, Survivor speaks out on ovarian cancer (Aug. 22, 1997) ("It is more prevalent in women who have had no pregnancies, have taken fertility drugs, had an early menopause, eaten a high-fat diet or frequently used talcum powder in the genital area.")

[15] Harvard Women's Health Watch, Ovarian cancer (Oct. 1998) ("Several studies also suggest that two other practices -- a high-fat diet and long-term use of talcum powder on the genital region -- increase the likelihood of ovarian cancer. Researchers theorize that talc travels into the vagina, cervix, uterus, and ultimately to the ovaries, where it may prompt cellular changes and, later, cancer.")

[16] Chicago Tribune, Talcum takes a tumble (July 14, 1999)

Appendix A: Sample Of Pre-2014 News Articles Addressing Posited Link Between Talc Use And Ovarian Cancer

[17] HealthCommunities.com, Ovarian Cancer Risk Factors, (August 14, 1999) ("Some research indicates that there is an increased risk of ovarian cancer among women who apply talcum powder to the genital area or sanitary napkins.")

[18] Cleveland Plain Dealer, Possible link between talcum powder, ovarian cancer (Aug. 17, 1999)

[19] CNN.com, Ovarian cancer: It's less common than you think (Sept. 3, 1999) ("The ruling on talcum powder is still unclear, as well. In the past, talcum powder was sometimes contaminated with asbestos, a known cancer-causing mineral.")

[20] Chicago Tribune, How much do you know about ovarian cancer? (Sept. 15, 1999) ("Some research has shown a possible link between talcum powders used in the genital region and an increased risk of ovarian cancer.")

[21] Las Vegas Review Journal, Some promising treatments being developed for ovarian cancer (Nov. 25, 1999) ("In support of this, scientists point to several studies showing that talcum powder, which some women put on diaphragms or on genital skin, can raise ovarian cancer risk.")

[22] Philadelphia Inquirer, Don't worry about talc in eye shadow, face powder (Jan. 16, 2000) ("That doesn't mean not to use eyeshadow or face powder with talc, but it absolutely means to consider never using it on your children, or vaginally on yourself.")

[23] St. Louis Dispatch, Can genital warts cause cancer? (Apr. 26, 2000) ("Talc, the main ingredient of talcum powder, has been linked to ovarian cancer when used as a vaginal dusting powder.")

[24] USA Today, Estrogen may join carcinogen list (Dec. 8, 2000) ("Research suggests that talcum powder used in feminine hygiene increases the risk of ovarian cancer.")

[25] New York Post, Feds eye new causes of cancer (Dec. 9, 2000) ("Meanwhile, talc has been linked to an increased risk of ovarian cancer in women who use it for feminine hygiene")

[26] Los Angeles Times, Study Suggests Aspirin May Help Prevent Ovarian Cancer (Mar. 12, 2001) ("Ovarian cancer might be preceded by inflammation due to pelvic inflammatory disease or the use of talcum powder, both of which are linked to an increased risk of the disease.")

[27] The Guardian, Is your beauty regime damaging your health? Once again, studies are suggesting that chemicals used in cosmetics such as talc could increase the risk of cancer. Just how worried should we be . . . (Sept. 11, 2007)

[28] National Health Service, Talcum powder and ovarian cancer (Sept. 29, 2008) ("Although this study has shortcomings and does not provide strong evidence of a causal link in itself, when put in context with other studies on this topic, it adds to the body of evidence suggesting that use of talc may be linked to ovarian cancer.")

[29] Washington Post, Cellphones are possible cancer risk, WHO says (June 1, 2011) ("Other substances that the group has categorized as 'possibly carcinogenic' include talcum powder, which has been possibly linked to ovarian cancer).

Appendix A: Sample Of Pre-2014 News Articles Addressing Posited Link Between Talc Use And Ovarian Cancer

[30] Cancer Weekly, Researchers from Pennsylvania State University Detail New Studies and Findings in the Area of Ovarian Cancer (Nov. 8, 2011) ("'A number of observational studies (largely case-control) conducted over the last two decades suggest an association between use of talc powders on the female perineum and increased risk of ovarian cancer")

[31] Women's Health Weekly, Recent findings from University of Queensland highlight research in ovarian cancer (Apr. 5, 2012) ("'Use of talcum powder in the perineal area has been associated with an increased risk of ovarian cancer")

[32] The Guardian, Ovarian cancer: a call to arms: Susan Gubar was when she was diagnosed with ovarian cancer. It wasn't exactly tragedy - her daughters fully grown, her work complete. The tragedy is the ignorance which still surrounds this neglected disease (Sept. 1, 2012) ("Asbestos exposure, talcum powder, hormone replacement therapy, and fallout from nuclear testing have all been linked to ovarian cancer")

[33] Huffington Post, Health Myths: 7 medical misconceptions exposed (May 16, 2013) ("Harvard researchers recently found that postmenopausal women who use talcum powder in their genital area just once a week increase their risk of developing endometrial cancer by 24 percent. Another Harvard study found a strong link between talcum powder use and ovarian cancer (it can increase the risk of developing the cancer by up to 40 percent.")

[34] Daily Mail, Women who regularly use talcum powder increase their risk of ovarian cancer by 24% (June 18, 2013) (The researchers analysed data from 8,525 women diagnosed with ovarian cancer and compared talcum powder use with that of 9,800 women who remained cancer-free. The results, published in the journal Cancer Prevention Research, showed regularly applying the powder particles after bathing or showering raised the risk of an ovarian tumour by 24 per cent.")

[35] Daily Mail (UK), Talc can raise ovarian cancer risk by quarter (June 19, 2013)

[36] The Sydney Morning Herald, Surprising cancer causes, (Aug. 02, 2013) ("Researchers have found a link between frequent use of talcum powder "for intimate personal hygiene" and ovarian cancer. The results published in the journal Cancer Prevention Research showed regularly applying the powder particles after bathing or showering raised the risk of an ovarian tumour by 24 per cent.")

[37] Rapid City Journal, South Dakota jury ties talc powder to cancer risk (Oct. 05, 2013) ("A federal jury in Sioux Falls has found that a woman's use of Johnson & Johnson products that contained talcum contributed to her ovarian cancer.")

[38] Reuters Legal, Johnson & Johnson failed to warn of possible talc-cancer link: jury (Oct. 8, 2013)

APPENDIX B

Materials Reviewed and Considered by Dr. Gregory Diette, M.D., M.H.S.

Expert References

Expert Report of Michael M. Crowley, Ph.D., Nov. 12, 2018 (MDL No. 2328)

Expert Report of William E. Longo, Ph.D, and Mark W. Rigler, Ph.D., Nov. 14, 2018

Expert Report of Sarah E. Kane, M.D., Nov. 15, 2018 (MDL No. 2738)

Expert Report of Rebecca Smith-Bindman, M.D., Nov. 15, 2019 (MDL No. 2738)

Expert Report of Alan Campion, Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Arch Carson, M.D., Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Daniel L. Clarke-Pearson, M.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Robert B. Cook, Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of David Kessler, M.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Mark Krekeler, Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Shawn Levy, Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Anne McTiernan, M.D., Ph.D., Nov. 16, 2019 (MDL No. 2738)

Expert Report of Patricia Moorman, M.S.P.H., Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Laura Plunkett , Ph.D., D.A.B.T., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Ghassan Saed, Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Sonal Singh, M.D., M.P.H., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Jack Siemiatycki, M.Sc., Ph.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Ellen Blair Smith, Nov. 16, 2018 (MDL No. 2738)

Expert Report of Judith Wolf, M.D., Nov. 16, 2018 (MDL No. 2738)

Expert Report of April Zambelli-Weiner, Ph.D., M.P.H., Nov. 16, 2018 (MDL No. 2738)

Expert Report of Judith Zelikoff, Ph.D., Nov. 16, 2018 (MDL No. 2738)

Deposition of Sonal Singh, M.D., M.P.H., Jan. 16, 2019 (MDL No. 2738)

Deposition of Anne McTiernan, Jan. 28, 2019 (MDL No. 2738)

Deposition of Patricia Moorman, M.S.P.H., Ph.D. Jan. 25, 2019 (MDL No. 2738)

Materials Reviewed and Considered by Dr. Gregory Diette, M.D., M.H.S.

Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738)

Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738)

Deposition of Jack Siemiatycki, Jan. 31, 2019 (MDL No. 2738)

Deposition of Rebecca Smith-Bindman, M.D., Feb. 7, 2019 (MDL No. 2738)

Deposition of Rebecca Smith-Bindman, M.D., Feb. 8, 2019 (MDL No. 2738)

Deposition of April Zambelli-Weiner, Ph.D., Jan. 11, 2019 (MDL No. 2738)

Deposition of April Zambelli-Weiner, Ph.D., Feb. 7, 2019 (MDL No. 2738)

Gynecologic Oncology Email dated Sept. 19, 2018 re: GYN-18-1020: Final Decision (Ex. 35 to the Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))

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APPENDIX C

CURRICULUM VITAE
The Johns Hopkins University School of Medicine

June 2017

GREGORY B. DIETTE, MD, MHS

DEMOGRAPHIC AND PERSONAL INFORMATION

Current Appointment:

University:

Professor of Medicine
Division of Pulmonary and Critical Care Medicine
The Johns Hopkins University School of Medicine
Baltimore, MD

Division of General Internal Medicine
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Education and Training

1981-1986	B.S.	The University of Pennsylvania Wharton School, Philadelphia, PA. BS degree in economics. Concentration: Management of Entrepreneurship
1981-1986	B.A.	The University of Pennsylvania School of Arts and Sciences Philadelphia, PA. English; Minor in Chemistry
1986-1990	M.D.	Temple University School of Medicine, Philadelphia, PA
1995-1997	M.H.S.	Johns Hopkins University, School of Hygiene and Public Health Epidemiology; Clinical Epidemiology

Post-Doctoral Training

1990-1993	Intern-Resident, Department of Internal Medicine, Hospital of the University of Pennsylvania, Philadelphia, PA
1994-1995	Clinical Fellow, Division of Pulmonary and Critical Care, Johns Hopkins University School of Medicine, Baltimore, MD
1995-1998	Research Fellow, Division of Pulmonary and Critical Care, Johns Hopkins University School of Medicine, Baltimore, MD

Professional Experience:

1991-1993	Assistant Clinical Instructor, University of Pennsylvania School of Medicine, Philadelphia, PA
1993-1994	Clinical Instructor, University of Pennsylvania School of Medicine, Philadelphia, PA
1993-1994	Attending Physician, Full-time, Department of Emergency Medicine, Hospital of the University of Pennsylvania, Philadelphia, PA
1996-1999	Senior Physician Scientist, Quality Assessment and Improvement Systems Division, Covance Health Economics and Outcomes Services. Washington, D.C.
1998-2000	Instructor, Departments of Medicine and Epidemiology, Johns Hopkins University, Baltimore, MD
1998-present	Attending Physician, Department of Medicine, Johns Hopkins Hospital and Johns Hopkins Bayview Medical Center, Baltimore, MD Duties include outpatient practice devoted to adult asthma and general pulmonary medicine; inpatient care in acute care hospital and intensive care units.
1998-2005	Core Faculty, Program for Medical Practice and Technology Assessment, Johns Hopkins University, Baltimore, MD

2000-2005 Assistant Professor of Medicine, Departments of Medicine and Epidemiology, Johns Hopkins University, Baltimore, MD

2005-2011 Associate Professor of Medicine, Departments of Medicine and Epidemiology, Johns Hopkins University, Baltimore, MD

2001-2015 Director of Clinical Research, Division of Pulmonary and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.

2011-Present Professor of Medicine, Schools of Medicine and Public Health, Johns Hopkins University, Baltimore, MD

2011-Present Director, Obstructive Lung Disease Program, Division of Pulmonary and Critical Care Medicine, Johns Hopkins University, Baltimore, MD

RESEARCH ACTIVITIES

Original Research

1. Grasso M, Weller WE, Shaffer TJ, **Diette GB**, Anderson GF. Capitation, Managed Care, and Chronic Obstructive Pulmonary Disease. American Journal of Respiratory and Critical Care Medicine 1998;158:133-138.

2. **Diette GB**, White P, Terry P, Jenckes M, Wise RA, Rubin H. Quality Assessment through Patient Self-Report of Symptoms Pre- and Post- Fiberoptic Bronchoscopy. Chest 1998;114:1446-1453.

3. **Diette GB**, Wiener CM, White P. The Higher Risk of Bleeding in Lung Transplant Recipients from Bronchoscopy is Independent of Traditional Bleeding Risks: Results of a Prospective Cohort Study. Chest 1999;115:397-402.

4. **Diette GB**, Wu AW, Skinner EA, Clark R, Markson L, McDonald R, Huber M, Markson L, Steinwachs D. Treatment Patterns Among Adult Asthmatics: Factors Associated with Overuse of Inhaled β-Agonists and Underuse of Inhaled Corticosteroids. Archives of Internal Medicine 1999;159: 2697-2704.

5. Barr LF, Campbell SE, **Diette GB**, Gabrielson EW, Kim S, Shim H, Dang CV. C-Myc Suppresses the Tumorigenicity of Lung Cancer Cells and Down-regulates Vascular Endothelial Growth Factor Expression. Cancer Research 2000; 60:143-9.

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EXTRAMURAL FUNDING:

Current Funding:

09/01/2015-08/31/2019

Obesity Enhances Susceptibility to Pollutant Effects in Asthma
NIH/NIEHS P50ES018176
Annual Direct: $1,051,797
PI: Hansel
Role: Co-Director, 25%
OBesity Enhances Susceptibility to Pollutant Effects in Asthma (OBESE ASTHMA), will study mechanisms by which obesity leads to enhanced susceptibility to pollutants (particulate matter with aerodynamic diameter < 2.5 μm (PM2.5) and ultrafine particles (UFP)) leading to increased asthma morbidity in children.

07/01/2015-06/30/2020

Comparing Urban and Rural Effects of Poverty on COPD (CURE COPD)
NIH/NIEHS P50ES026096
Annual Direct: $693,432
PI: Hansel
Role: Co-Director, 5%
Comparing Urban and Rural Effects of Poverty on COPD (CURE COPD) Annual Direct Cost: $693,432 Principal Investigator, 23% effort The aim of our Center, Comparing Urban and Rural Effects of poverty on COPD (CURE COPD), is to understand these interactive effects (high indoor air pollution, obesity and pro-inflammatory diets) in both urban (Project 1) and rural (Project 2) low income communities, both of which suffer disproportionate prevalence and morbidity from COPD.

09/01/2012-08/31/2017

K-24 Mentoring and Patient Oriented Research in Asthma
NIH/NIEHS K24ES021098
Annual Direct: $182,540.00
Grant Number 1133451
PI: Diette
Role: Principal Investigator, 6.0 calendar months
A major focus of this proposal will be to expand the present research program from inner city children to also include inner city adults with asthma. With this expansion in the research program, the candidate will provide the foundation for future trials in adults of home-based multi-component environmental interventions, goals which are concordant with the career goals of current mentees and will establish the infrastructure for future mentees with a research interest in adult asthma

09/01/2009-07/31/2015

Title: Mechanisms of asthma-dietary interventions against environmental triggers (No cost extension)
P01 ES018176
NIH/NIEHS/EPA
Total Direct $4,999,821 ($970,685 Year 3)
PI/PD: Diette
Roles: Program Director, Administrative Core Leader, Project 1 Leader. 3.0 calendar months

Goals: The long-term goal of the **ASTHMA-DIET** (**A S**tudy to understand **Th**e **M**echanisms of **A**sthma--**D**ietary **I**nterventions to protect against **E**nvironmental **T**riggers) Program is to understand how diet influences the asthmatic response to indoor and outdoor airborne pollutants and allergens, with the expectation of translating these findings into practical dietary strategies to improve pediatric asthma health.

09/07/2010-04/30/2015

Genetic susceptibility to asthma and indoor air pollution in Peru
R01 ES018845
NIH/NIEHS
Annual Direct Cost: $433,836
PI: Hansel
Role: Co-Investigator, 1.20 calendar months
The goal of this proposal is to examine the contribution of genetic susceptibility to the adverse effects of indoor air pollution (particulate matter and nitrogen dioxide) on asthma health in a Hispanic population.

02/18/2011-12/31/2015

Statistical methods for complex environmental health data.
R01 ES019560
NIH/NIEHS
Annual Direct: $243,746
PI: Peng
Role: Co-Investigator, 0.60 calendar months
This project will develop a spatial-temporal Bayesian hierarchical multivariate receptor model for identifying sources of air pollution chemical mixtures and estimating their effect on population health outcomes. Innovation focuses on (a) conducting an integrated national assessment of the health effects of pollution sources; (b) the use of spatial-temporal models for source apportionment; and (c) the introduction of national databases on source profiles and emissions to inform model development and parameter estimation. These methods will be applied to data from a national study of air pollution and health outcomes, the Medicare Cohort Air Pollution Study, to (a) estimate short-term population health effects of PM sources on a national, regional, and local scale; (b) estimate short- and long-term health effects of PM constituents and identify the sources of toxic constituents

PAST (most recent 5 years only)

09/14/2007-08/31/2013 (NCE)

SCCOR: Mechanisms and Treatment of COPD Progression
1P50HL084945-01
NIH/NHLBI
Annual Direct $1,957,399
Program Director: Wise
Role: Core C Leader, 1.2 calendar months
The overall goal of this SCCOR program is to understand the complex interplay of mechanisms that promote the progression of COPD and to translate that understanding into treatments that can benefit persons who suffer from COPD.

09/29/2007-06/30/2013

Center for Childhood Asthma in the Urban Environment

(NCE)	The Role of Particulate Matter and Allergens in Oxidative Stress in Asthma (DISCOVER) 1P50ES015903 NIH/NIEHS Annual Direct $ 1,607,733 PI/PD: Breysse Roles: Co-Program Director; Project Leader, Project 1 (1.2 calendar months); Co-Investigator, Administrative Core (1.8 calendar months) The long-term goals of this Center are to examine how exposures to environmental pollutants and allergens may relate to airway inflammation and respiratory morbidity in children with asthma living in the inner city of Baltimore, and to search for new ways to reduce asthma morbidity by reducing exposure to these agents.
07/01/2008-06/30/2013	The Impact of Indoor Particulate Matter Exposure on Non-allergic Asthma 5K23 ES016819 NIH/NIEHS Total Direct: $755,875 PI: McCormack Role: Mentor, no salary support K23 Mentored Patient-Oriented Research Career Development Award The goal of this project is to examine adverse effects of coarse indoor PM. Using a study design that combines a longitudinal panel study and an exposure challenge model the research will demonstrate a causal relationship between indoor coarse PM exposure and exacerbation of asthma status.
07/01/2010-06/30/2012	Vitamin D and Susceptibility to Inhaled Pollutants in Urban Children with Asthma NIH Total Direct: $187,645 PI: Bose Role: Primary Mentor NRSA. The goal of this study is to identify the role of vitamin D upon the effects of inhaled pollutants upon asthma severity in inner-city children.
07/01/2011-09/24/2012	Interventions to Modify Adherence to Asthma Guidelines HHSA 290 2007 10061 I Agency: AHRQ Annual Direct Costs: $260,643 PI: Eric Bass Role: Co-PI The objective of this CER is to determine the comparative effectiveness of interventions to modify the adherence of health care providers to asthma guidelines.
12/15/2008-12/14/2011	Intervention trial to reduce nitrogen dioxide and carbon monoxide concentrations in Baltimore City homes FR-5200-N-01A HUD Annual Direct $271,415

PI: Hansel
Role: Co-Investigator, 0.96 calendar months
The purpose of this research is to conduct a randomized intervention trial aimed at reducing indoor nitrogen dioxide and carbon monoxide concentrations in homes.

07/08/2009-06/30/2011

Effect of Fenzian treatment on symptoms, pulmonary function and Albuterol use in patients with mild persistent asthma: A multi-center, sham-controlled clinical trial
Fenzian, Inc. (Formerly Eumedics)
Annual Direct: $88,433
PI: Diette, 1.20 calendar months
The purpose of the study is to test the efficacy of Fenzian treatment over five weeks to improve asthma control, pulmonary function, symptoms and bronchodilator use.

07/01/2006-06/30/2011

Mouse Allergen and Inner-City Asthma
1R01 A1070630-01
NIH/NIAAD
Annual Direct $225,000
PI: Matsui
Role: Co-Investigator, 0.60 calendar months
The primary aims of this project are (1) to examine the link between household mouse allergen exposure and asthma morbidity, and (2) to determine the diagnostic utility of allergy skin testing in predicting allergic airways responses to mouse allergen.

12/26/2003-01/30/2011

Evaluation of home automated tele-management in COPD.
R01 AI070630
NIH
Annual Direct: $225,000
PI: Finkelstein
Role: Co-Investigator, 0.60 calendar months
The goal of this project is to evaluate the impact of home tele-management in COPD patients.

12/01/2005-11/30/2010

A Multicenter Randomized Clinical Trial: Asthma Intervention Research (AIR2 Trial)
Asthmatix, Inc
Annual Direct $313,504
PI: Yung
Role: Co-Investigator, 0.12 calendar months
The goal of this trial is to assess the safety and effectiveness of the Alair system for the treatment of asthma.

11/1/2003-10/31/2009

Center for Childhood Asthma in the Urban Environment
P01 R-826724/P01 ES09606 (Breysse)
NIH/EHS/EPA
Annual Direct $918,780
PI: Breysse

Roles: Deputy Program Director; PI of Epidemiology Component, Co-Investigator, 1.5 calendar months
The long term goals of this Center were to examine how exposures to environmental pollutants and allergens might relate to airway inflammation and respiratory morbidity in children with asthma living in the inner city of Baltimore, and to search for new ways to reduce asthma morbidity by reducing exposure to these agents.

09/30/2003-06/30/2009

SCCOR: Ventilator associated lung injury: Molecular approaches
P50 HL073944-03 (Brower)
NIH/NHLBI
Annual Direct $2,790,934
PI/PD: Brower
Role: Core Leader, Core B, Data Management Core, 0.60 calendar months
This SCCOR was focused on understanding the complex interplay between mechanical ventilation and the increased morbidity and mortality associated with acute lung injury. The application had interactive Cores using state of the art approaches to provide understanding of critical pathobiologic processes in ventilator-associated lung injury and to define key genetic determinants relevant to acute lung injury.

09/30/2004-08/31/2009

Genetics of Asthma Severity and Lung Function Decline
K23 HL76322 -02
NIH/NHLBI
Annual Direct $148,250
PI: Hansel
Role: Primary Mentor, effort as needed
The goal of this study was to identify genetic polymorphisms that mark high risk individuals for early intervention to decrease asthma morbidity.

09/10/2001-08/31/2006

Improving physician adherence to asthma guidelines
K23 HL04266
NIH
Annual Direct $146,772
Role: Principal Investigator, 9.0 calendar months
Provide mentored training and research period for early career development. Improve physician adherence to national asthma guidelines

09/01/2002-07/31/2007

Baltimore Asthma Severity Study
R01 HL67905 (Ford**)**
NIH
Annual Direct $443,417
PI: Ford
Role: Co-Investigator, 0.6 calendar months
The objective of this study was to provide insight into the genes controlling susceptibility to human asthma and promote the development of novel therapeutics.

9/01/2002-08/31/2007

Improving Respiratory Outcomes in ALS

	K23 HL67887 (Lechtzin) NIH Annual Direct $121,750 PI: Lechtzin Role: Advisor (effort as needed) The overall theme of this award is to study various aspects of non-invasive positive pressure ventilation in patients with ALS with the goal of improving respiratory management of these patients.
2007-2008 (NCE)	Howard/Hopkins Center for Reducing Asthma Disparities HL072455 NIH/NHLBI Annual Direct $513,475 PI: Rand Role: Leader, Project 1, 1.5 calendar months, no cost extension This application presents four research projects designed to collaboratively investigate factors associated with the disproportionate burden of asthma experienced by inner-city, African-American children and adults.
09/30/2004-06/30/2008	Improving asthma care for minority children in Head Start R18 HL73833 NIH Annual Direct $625,506 PI: Rand Role: Co-Investigator, 0.6 calendar months The goal of this project is to study the effect communication intervention on asthma-related morbidity and mortality among low-income African American children.
02/23/2004-12/31/2006	A randomized, sham-controlled, double-blinded pilot study to assess the effect of high frequency chest wall oscillation therapy in patients with chronic bronchitis Advanced Respiratory PI: Diette, 0.12 calendar months
10/01/2007-09/30/2009	Randomized clinical trial Protocol #: CQAB149B2335S Novartis Total Direct Costs: $161,368 PI: Diette, 1.20 calendar months A 26-week treatment, multicenter, randomized, double-blind, double dummy, placebo-controlled, adaptive, seamless, parallel-group study to assess the efficacy, safety and tolerability of two doses of indacaterol (selected from 75, 150, 300 & 600 ug o.d.) in patients with chronic obstructive pulmonary disease using blinded formoterol (12 ug b.i.d.) and open label tiotropium (18 ug o.d.) as active controls.

EDUCATIONAL ACTIVITIES

Educational Publications

Invited Review Articles

1. Rubinson L, Diette GB. Best Practices for Insertion of Central Venous Catheters in Intensive Care Units to Prevent Catheter-Related Bloodstream Infections. Journal of Laboratory and Clinical Medicine 2004;143:5-13.

2. Sharma HP, Hansel NN, Matsui EC, Diette GB, Eggleston PA, Breysee PN. Indoor Environmental Influences on Children's Asthma. Pediatric Clinics North America. 2007;54:103-120

3. Hansel NN and Diette GB. Gene Expression Profiling in Human Asthma. Proc Am Thorac Soc. 2007; 4(1):32-6.

4. **Diette GB**, Rand C. The Contributing Role of Health-Care Communication to Health Disparities for Minority Patients with Asthma. Chest. 2007 Nov;132(5 Suppl):802S-9S.

5. **Diette GB**, McCormack MC, Hansel NN, Breysee PN, Matsui EC. Environmental issues in managing asthma. Respiratory Care. 2008;53(5):602-15; discussion 616-7.

6. Matsui EC, Hansel NN, McCormack MC, Rusher R, Breysse P, **Diette GB**. Asthma in the Inner City and the Indoor Environment. Immunology Allergy Clinics North America. 2008;28:665-686.

7. Okelo SO, Butz AM, Sharma R, **Diette GB**, Pitts SI, King TM, Linn ST, Reuben M. Chelladurai Y, Robinson KA. Interventions to modify health care provider adherence to asthma guidelines: A systemic review. Pediatrics. 2013 Sep;132(3):517-34.

Editorials

1. Krishnan JA, **Diette GB**, Rand CS. Disparities in Outcomes from Chronic Disease: Impaired Patient-Physician Partnerships May Be an Important Cause in Minorities. British Medical Journal 2001;323:950.

2. Alberg A, **Diette GB**, Ford J. Attendance and absence as markers of health status: The example of active and passive cigarette smoking. American Journal of Epidemiology 2003 May 15;157(10):870-3.

3. **Diette GB**, Clinical Commentary: Overuse of β2-agonists. *J Resp Diseases* 2000;21:721.

Case Reports

None.

Letters

1. Patil S**,** Krishnan JA, Lechtzin N, **Diette GB**. In-hospital mortality following acute exacerbation of chronic obstructive pulmonary disease. *Archives of Internal Medicine*. 2004 Jan 26;164:222-223.

2. **Diette GB**, Wu AW. Elderly asthmatic patients. *Archives of Internal Medicine*. 2003 Jan 13;163;1:122.

4. Clerisme-Beaty EM, Rand C, **Diette GB.** Reply to Farah. Weight loss in asthma: More evidence is needed. Reply to Farah.Journal of Allergy and Clinical Immunology 2010 125(3):770. PMCID: PMC2908807.

Book Chapters:

1. **Diette G**, Brower R. Traditional Invasive Ventilation. In, Pulmonary Respiratory Therapy Secrets, 2nd Edition, Parsons P and Heffner J, Eds., Philadelphia, Hanley & Belfus, 2002.

2. **Diette G**, Brower R. Traditional Invasive Ventilation. In, Pulmonary Respiratory Therapy Secrets, Parsons P and Heffner J, Eds., Philadelphia, Hanley & Belfus, 1997.

3. **Diette G.** Pleural Effusion. In, Mosby's Success in Medicine Specialty Clinical Sciences, Donnelly JL, Ed., Mosby, 1996.

5. **Diette G.** Pneumothorax. In, Mosby's Success in Medicine Specialty Clinical Sciences, Donnelly JL, Ed., Mosby, 1996.

6. **Bose S, Diette GB.** Health disparities related to environmental air quality. In: Health Disparities in Respiratory Medicine. Eds: Gerald L and Berry C. Springer. In press.

Internet:

Diette GB, Liu MC. Disease Update on Asthma. Medcast Networks. [Released March 1, 1999]

Okelo SO, Butz AM, Sharma R, **Diette GB**, Pitts SI, King TM, Linn ST, Reuben M, Chelladurai Y, Robinson KA. Interventions to modify health care provider adherence to asthma guidelines [Internet]. Rockville MD: Agency for Healthcare Research and Quality (US); 2013 May.

Reports:

1. Wu A, **Diette GB**, Skinner E, Clark R, Steinwachs D. Treatment Patterns Among Adult Asthmatics: Factors Associated with High Use of Inhaled β-agonists, Low Use of Inhaled Corticosteroids, and Nocturnal Symptoms. Submitted to Merck & Co., Inc., July 1997.

2. Steinberg EP, Holtz PM, Greenwald TP, **Diette GB**, Wills S, Webb A, Daugherty L, Caravoulias CL, Gabrielsen M, Pomponio C. Report of results of a pilot test of draft NCQA HEDIS measures of health plan performance in control of blood pressure among diagnosed hypertensives. Submitted to the NCQA Hypertension Measure Advisory Committee, July 1998.

3. Wu AW, Skinner EA, **Diette GB**, Nguyen TTH, Clark RD. Quality of Care and Outcomes for Childhood Asthma in Managed Care: Validation of the Asthma Therapy Assessment Questionnaire. Submitted to Merck & Co., Inc., November 1998.

4. **Diette GB**, Krishnan JA, Lechtzin N, Belcastro D. Evidence Report on Chronic Obstructive Pulmonary Disease: Treatment and Risks. Submitted to CardioContinuum, September 1999.

5. Wu AW, **Diette GB**, Dominici F, Skinner EA. The 1998 Asthma Outcomes Survey: Phase I Final Report. Submitted to the Pacific Business Group on Health, October 1999.

6. **Diette GB**, Krishnan JA, Lechtzin N, Belcastro D. Report on Focus Group of Clinician Experts on Treatment of Chronic Obstructive Pulmonary Disease. Submitted to CardioContinuum, September 1999.

7. **Diette GB,** Qutami M, Sullivan B. Report on Cystic Fibrosis Utilization and Asthma Utilization and Medication Use. Submitted to Aerogen, December 1999.

8. **Diette GB**, Rand C, Wise RA, Thompson K, Merriman B. Pilot Study of Alternative Treatment Settings of High Frequency Chest Wall Oscillation in Patients with Chronic Bronchitis. Submitted to Advanced Respiratory, Inc, December 2003.

Teaching

Classroom Instruction

1993-1994	Instructor, Course on Clinical Management in the Emergency Department, University of Pennsylvania Department of Emergency Medicine
12/1993	Instructor, First Aid for First Year Medical Students, University of Pennsylvania School of Medicine
1996 & 1999	Clinical Faculty for Human Anatomy Discussion Group, Heart and Lungs, Johns Hopkins University School of Medicine
1997-2000	Instructor, Evidence-Based Medicine Rotation for Medical Interns, Chronic Obstructive Pulmonary Diseases, Department of Medicine, Johns Hopkins Bayview Medical Center
1997 & 2000	Discussion Leader, Organ Systems Course, Pulmonary Physiology Section, Johns Hopkins University School of Medicine
1997	Teaching Assistant, The Science of Clinical Investigation: Design of Clinical Studies. Johns Hopkins University School of Hygiene and Public Health
1998	Lecturer, Clinical Skills Course: The Pulmonary Examination, Johns Hopkins University School of Medicine
1999 & 2000	Discussion Leader, Pathophysiology Course, Pathophysiology of Shock, Johns Hopkins University School of Medicine
1999	Lecturer, Advanced Research Methods, International Respiratory Epidemiology Course, American Thoracic Society, Cusco, Peru
1999-2002	Co-Director. The Science of Clinical Investigation: Design of Clinical Studies. Johns Hopkins University School of Hygiene and Public Health
2000-2003	Lecturer, Patient Outcomes and Quality of Care Course, Department of Health Policy and Management, Johns Hopkins University School of Hygiene and Public Health
2000	Lecturer, Advance Research Methods, International Respiratory Epidemiology Course,

American Thoracic Society, Quinamavida, Chile

2001 Discussion Leader, Clinical Epidemiology, Department of Epidemiology, Johns Hopkins University, April 2001.

2003 Co-Director. Advanced Research Methods, International Respiratory Epidemiology Course, American Thoracic Society, Buenos Aires, Argentine

2004 Director. Advanced Research Methods, Method in Epidemiologic, Clinical and Operations Research, American Thoracic Society, Punta del Este, Uruguay

2005 Faculty. Methods in Clinical Research. ERS/ATS School Course. Prague, Czech Republic,

2005 Director. Advanced Research Methods, Methods in Epidemiologic, Clinical and Operations Research, American Thoracic Society, Quito, Ecuador

2006 Director. Advanced Research Methods, Methods in Epidemiologic, Clinical and Operations Research, American Thoracic Society, Alphaville, Brazil.

2007-Present Attending Physician, the Barker Firm, Johns Hopkins University School of Medicine

Continuing Medical Education

2010 Managed care strategies used in the successful treatment of asthma. National Asthma Education and Prevention Program. Medical Communications Media, Inc.

Mentoring (pre- and post-doctoral):

Advisees

2012-Present Emily Bingham, MD
Post-doctoral Fellow, Division of Pulmonary and Critical Care Medicine

2011-Present Laura M. Paulin, MD
Post-doctoral Fellow, Division of Pulmonary and Critical Care Medicine

2012-Present Jessica Rice, MD
Post-doctoral Fellow, Department of Pediatrics

2010-2014 Niru Putcha, MD
Post-doctoral Fellow, Division of Pulmonary and Critical Care Medicine

2010-2011 Daniel Jamieson, MD
Post-doctoral Fellow, Division of Pulmonary and Critical Care Medicine

2009-Present Sonali Bose, MD, MPH
Post-doctoral Fellow, Division of Pulmonary and Critical Care Medicine
Research Theme: Vitamin D levels in urban black children with asthma

Current Position: Instructor of Medicine, Pulmonary and Critical Care Medicine

2009-2010 Marisha Cook, MD
Post-doctoral Fellow, Division of Allergy and Clinical Immunology
Research Theme: Dietary pattern differences by race in asthma
Current Position: Post-doctoral Fellow, Allergy & Clinical Immunology

2008-2009 Timothy Scialla, MD
Post-doctoral Fellow, Division of Pulmonary and Critical Care Medicine
Research Theme: Inner City Diet and Asthma
Current Position: Assistant Professor of Medicine, University of Miami, Miami, Florida

2006-2007 Sabine Karem, MD
Post-doctoral Fellow, Division of Pulmonary and Critical Care Medicine
Research Theme: Asthma Control in African-Americans
Current Position: Internal Medicine Resident, Montifiore Hospital, Bronx, NY

2005-2006 Lindsey Kim
MPH student, School of Hygiene and Public Health
Thesis: Outcomes Study on Environmental Control Practices on Health of Inner-City Children with Asthma

2005-2007 Emily Smith Tonorezos
Post-Doctoral Fellow, Division of General Internal Medicine
Research Theme: Diabetes as a modifying factor on the effect of particulate matter in COPD
Current Position: Assistant Professor of Medicine, Memorial Sloan Kettering, New York.

2005-2008 Meredith C. McCormack, MD, MHS
Post-Doctoral Fellow, Division of Pulmonary and Critical Care Medicine
Awarded Chest Foundation Award for Women's Studies, Loan Repayment Program, NIH K-23 Award, Johns Hopkins Bloomberg School of Public Health Faculty Grant in Global Health, and Pearl M. Stetler Research Fund
Research Theme: Particulate Matter Effects on Asthma and COPD
Current Position: Assistant Professor of Medicine, Johns Hopkins University, Baltimore, Maryland

2004-2006 Amit Rahman
Medical Student, Johns Hopkins University, School of Medicine
Research Theme: Co-Morbidity COPD Outcomes

2004-2005 Alan Salas
Under-represented Minority Summer Research Program
Undergraduate Student, Johns Hopkins University, Baltimore, MD
Research Theme: Early Life Exposures and Risk of Asthma

2002-2003 Deanna Perez Williams
Community Health Scholars Program, Kellogg Foundation

Research Theme: Development of a Culturally-Sensitive, Patient-Focused Asthma Communication Instrument Designed to Enhance Provider-Patient Communication in Hispanics in Baltimore
Current Position: Howard University

2002-2004 Elizabeth C. Matsui, MD
Post-Doctoral Fellow, Division of Allergy and Immunology, Department of Pediatrics, Johns Hopkins University
Research Theme: Mouse allergen exposure, antibody responses, prick skin test response and allergy symptoms in laboratory workers
Current Position: Associate Professor of Pediatrics. Division of Allergy and Immunology, Department of Pediatrics, Johns Hopkins University

2002-2004 Necole Streeper, MD
Minority Summer Research Program
Research Theme: Physician Underestimation of Self-Management Ability of African-Americans with Asthma
Current Position: Resident, Dept of Urology, University of Texas HSC, San Antonio, TX

2002-2004 James Lee, MD
Housestaff, Internal Medicine, Johns Hopkins Hospital
Research Theme: Gender Differences in Childhood Asthma
Current Position: Assistant Professor of Medicine, Division of Pulmonary, Allergy and Critical Care Medicine, Hospital of the University of Pennsylvania, Philadelphia, PA

2002-2007 Cecilia Patino, MD
Research Associate, Division of Pulmonary and Critical Care Medicine
Research Theme: (1) Physician Adherence to Asthma Guidelines; (2) Validation of Survey Methods of Environmental Assessment
Current Position: Assistant Professor, Department of Preventive Medicine, University of Southern California, Los Angeles, CA

2001-2003 Marianelle Platon, MD
Under-represented Minority Summer Research Program
Research Theme: Validation of Physician Reported Adverse Events during Bronchoscopy
Current Position: Physician, National Navel Medical Center, Bethesda, Maryland

2001-2004 Lucian Davis, MD
Housestaff, Internal Medicine, Johns Hopkins Hospital
Research Theme: Predictors of New-Onset Dyspnea in COPD
Current Position: Assistant Adjunct Professor, Division of Pulmonary and Critical Care Medicine, University of California, San Francisco, San Francisco.

2001-2005 Susan Gerhardt, MD
Post-Doctoral Fellow, Division of Pulmonary and Critical Care Medicine
Awarded Pearl M. Stetler Research Grant
Research Theme: Treatment of Bronchiolitis Obliterans in Lung Transplant Rejection
Current Position: Private Practice, Pennsylvania

2000-2005 Lewis J. Rubinson, MD

Post-Doctoral fellow, Division of Pulmonary and Critical Care Medicine
Research theme: National Guidelines and Central Venous Catheter Infections in the Intensive Care Unit
Current Position: Assistant Professor, Division of Pulmonary and Critical Care Medicine, University of Washington, Seattle.

2000-2003 Sande Okelo, MD
Post-Doctoral fellow, Division of Pediatric Pulmonary Medicine
Research theme: Emotional Function and Asthma Morbidity in Children
Awarded NIEHS Minority Supplement Award
Awarded ATS Minority Travel Award
Current Position: Assistant Professor, Department of Pediatrics, David Geffen School of Medicine at UCLA, Mattel Children's Hospital UCLA, Los Angeles, CA

2000-2004 Nadia N. Hansel, MD, MHS
Post-Doctoral fellow, Division of Pulmonary and Critical Care Medicine
Awarded Howard C. and Jane R. Goodman Award
Awarded the Baurernschmidt Fellowship Award from Eudowood Foundation
Awarded Chest Foundation Award for Women's Studies
Awarded American Thoracic Society Underrepresented Minority Travel Award
Research themes: 1) Quality of Life in Tuberculosis; 2) Th1/Th2 phenotype in tuberculosis and asthma.
Current Position: Associate Professor of Medicine, Johns Hopkins University

1999-2001 Edward Cox, Jr., MD, MPH
MPH student, School of Hygiene and Public Health
Project: Association of Hospital Volume and In-Hospital Mortality among Patients with Community-Acquired Pneumonia
Current Position: Director, Office of Antimicrobial Products (OAP) Food and Drug Administration, Rockville, Maryland

1999-2001 Noah Lechtzin, MD, MPH
Post-Doctoral fellow, Division of Pulmonary and Critical Care Medicine
Awarded Travel Award for Poster Presentation at 2001 American Thoracic Society International Meeting
Research theme: Respiratory manifestations of ALS: 1. Measures of disease burden; 2. Improving patient outcomes.
Current Position: Associate Professor of Medicine, Johns Hopkins University.

1998-2001 Jerry A. Krishnan, MD, PhD
Post-Doctoral fellow, Division of Pulmonary and Critical Care Medicine.
Awarded Chest Foundation Research Award for "Assessment of Gender and Race Differences in Quality of Care and Clinical Outcomes from Asthma."
Research theme: Quality of care and outcomes for asthma by gender and race
Current Position: Professor of Medicine, University of Illinois, Chicago.

1998-1999 Su Wang
MPH student, School of Hygiene and Public Health

Thesis: Nocturnal Symptoms in Pediatric Asthma: Clinical Features and Health Care Utilization in a Managed Care Setting
Current Position: Unknown.

1997-2002 Lindy Wolfenden, MD
Housestaff, Internal Medicine, Johns Hopkins Hospital
Post-Doctoral Fellow, Division of Pulmonary and Critical Care Medicine
Research Theme: Older Adults and Asthma
(Deceased.)

Thesis committees

07/2014 Kamau Peters, Doctoral Candidate in Environmental Health Scoenvces.
Role: Thesis Advisor and Final Oral Examination Committee Member.

04/2013 María Fernanda Cely-García, Doctoral Candidate, Universidad de Los Andes, Bogotá Columbia (*Personal exposures to asbestos and respiratory health of automotive mechanics in Bogotá, Columbia*)
Role: Thesis advisor and Final Oral Defense Committee Member

04/2010 Deanna M. Green, Doctoral Candidate in Environmental Health Sciences
Role: Thesis Advisor and Final Oral Defense Committee Member

10/2008 Maura Dwyer, Doctoral Candidate in Environmental Health Sciences
Role: Final Oral Defense Committee Member

10/2007 Juan Ramos Bonilla, Doctoral Candidate in Environmental Health Sciences
Role: Final Oral Defense Committee Member

12/2006 Sorina Eftin, Doctoral Candidate in Environmental Health Sciences
Role: Thesis Committee Chair

04/2005 Laura LaRosa, Doctoral Candidate in Environmental Health Engineering
Role: Final Oral Defense Committee Member

11/2005 Kannika Taenkhum, Doctoral Candidate in Environmental Health Engineering
Role: Preliminary Orals Committee Member

12/2005 Sande Okele, Doctoral Candidate in Graduate Training Program in Clinical Investigation
Role: Final Oral Defense Committee Member

09/2004 Lewis Rubinson, Doctoral Candidate in Epidemiology
Role: Final Oral Defense Committee Member

03/2003 Ichan Huang, Doctoral Candidate in Health Policy and Management
Role: Thesis Committee Chair

03/2002 Ichan Huang, Doctoral Candidate in Health Policy and Management
Role: Preliminary Orals Committee Member

10/2001 Erika Tang, Doctoral Candidate in Epidemiology
Role: Preliminary Orals Committee Member

Editorial Activities

Peer review activities

Editorial Boards

2010- Present Member, *Clinical Respiratory Journal*
2013-Present Member, *Journal of Pollution Effects & Control*

Peer Reviewer

American Journal of Respiratory and Critical Care Medicine
Archives of Internal Medicine
Archives of Pediatric and Adolescent Medicine
Cancer Epidemiology, Biomarkers & Prevention
Chest
Epidemiology
Expert Opinion on Pharmacotherapy
Health Services Research
Journal of Allergy and Clinical Immunology
Journal of Clinical Outcomes Management
Journal of General Internal Medicine
Journal of Respiratory Diseases
Medical Care
Pediatrics
Preventative Medicine in Managed Care
Quality of Life Research
Thorax

CLINICAL ACTIVITIES:

Certification:

MEDICAL LICENSURE Maryland D-47616

BOARD CERTIFICATION

1991	National Board of Medical Examiners
1993	American Board of Internal Medicine
1996, 2006	American Board of Internal Medicine, Pulmonary Medicine

Service Responsibilities (specialty, role, time commitment):

Intensive Care Medicine, Attending Physician,
Oncology Center, Pulmonary and Critical Care Service, Attending Physician
Pulmonary Inpatient Medicine, Attending Physician
Barker Inpatient Internal Medicine, Attending Physician
Outpatient Pulmonary Clinic, Attending Physician

SYSTEM INNOVATION AND QUALITY IMPROVEMENT ACTIVITIES

System Innovation and Quality Improvement Publications

Please see original research citation numbers 2, 3, 4, 6, 7, 9, 11, 12, 13, 14, 15, 17, 20, 21, 22, 23, 24, 26, 27, 29, 30, 31, 34, 36, 42, 43, 44, 45, 47, 52, 59, 68, 69, 70, 73, 75,76, 81, 84, 88, 89, 92, 93, 95 and 99.

System Innovation and Quality Improvement efforts within JHM:

1996-2006 **Initiator and Director**, Bronchoscopy Quality Improvement Project (BRONCHQI), Johns Hopkins Medical Institutions, Baltimore, MD

This highly successful project had many findings, including:

1. Documentation of unsafe dosing of lidocaine, which led to a reduction in the strength used from 2% to 1%. Documented no loss of analgesia with the change.
2. Identified risk of bleeding complications with lung biopsy
3. Documented diagnostic utility of having on-site cytopathology services during needle biopsy cases
4. Identified factors associated with patient satisfaction
5. Identified excessive pain and reasons for pain during the procedure
6. Performed a clinical trial of distraction therapy to reduce pain during the procedure
7. Identified predictors of positive diagnostic findings in immune-compromised patients
8. Demonstrated benefits of use of atropine pre-procedure to prevent adverse events

1997-2000 **Member**, Committee for Procedure Review, Pulmonary and Critical Care Medicine Procedures, Johns Hopkins Bayview Medical Center, Baltimore, MD

System Innovation and Quality Improvement efforts outside JHM:

1996-1999 Senior Physician Scientist, Quality Assessment and Improvement Systems Division, Covance Health Economics and Outcomes Services. Washington, D.C.

Dialysis Outcomes Quality Initiative (DOQI): Co-investigator, Medical consultant, NCQA HEDIS hypertension measure: Co-investigator on measure validation

2003 **Member,** Howard County Comprehensive Health Improvement Plan for the Year 2010, Howard County Health Department, Columbia, MD

National Committee for Quality Assurance

2003 Member, COPD Technical Subgroup
2004-Present Member, Clinical Expert Panel
2008 Member, National Committee for Quality Assurance (NCQA) Advisory Panel. HEDIS Trends Publication Expert Advisory Panel.

Production of guidelines and/or protocols:

2002 American Healthways/Johns Hopkins
2nd Annual Disease Management Outcomes Summit: Standard Outcome Metrics and Evaluation Methodology for Disease Management Programs, November 7-10, 2002, Palm Desert, CA.
Role: Physician Steering Committee. The outcome metrics remain intact to date.

System Innovation and Quality Improvement Program Building/Leadership:

N/A

System Innovation and Quality Improvement Extramural Funding

12/26/2003-01/30/2011 Evaluation of home automated tele-management in COPD.
R01 AI070630
NIH
Annual Direct: $225,000
PI: Finkelstein

Role: Co-Investigator, 0.60 calendar months

09/10/2001-08/31/2006
Improving physician adherence to asthma guidelines
K23 HL04266
NIH
Annual Direct $146,772
Role: Principal Investigator, 9.0 calendar months
Provide mentored training and research period for early career development.
Improve physician adherence to national asthma guidelines

9/01/2002-08/31/2007
Improving Respiratory Outcomes in ALS
K23 HL67887 (Lechtzin)
NIH
Annual Direct $121,750
PI: Lechtzin
Role: Advisor (effort as needed)
The overall theme of this award is to study various aspects of non-invasive positive pressure ventilation in patients with ALS with the goal of improving respiratory management of these patients.

2007-2008 (NCE)
Howard/Hopkins Center for Reducing Asthma Disparities
HL072455
NIH/NHLBI
Annual Direct $513,475
PI: Rand
Role: Leader, Project 1, 1.5 calendar months, no cost extension
This application presents four research projects designed to collaboratively investigate factors associated with the disproportionate burden of asthma experienced by inner-city, African-American children and adults.

09/30/2004-06/30/2008
Improving asthma care for minority children in Head Start
R18 HL73833
NIH
Annual Direct $625,506
PI: Rand
Role: Co-Investigator, 0.6 calendar months
The goal of this project is to study the effect communication intervention on asthma-related morbidity and mortality among low-income African American children.

ORGANIZATIONAL ACTIVITIES

Institutional Administrative Appointments

1995-1997 **Initiator and Coordinator**, Pulmonary and Critical Care Epidemiology Seminar, Johns Hopkins University, Baltimore, MD

1996-present **Initiator and Director**, Bronchoscopy Quality Improvement Project (BRONCHQI), Johns Hopkins Medical Institutions, Baltimore, MD

1997-2000 **Member**, Committee for Procedure Review, Pulmonary and

Critical Care Medicine Procedures, Johns Hopkins Bayview Medical Center, Baltimore, MD

1999-present **Member,** Education Committee, Pulmonary and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD

1999-present **Member,** Research Committee, Pulmonary and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD

1999-2006 **Chair,** Conference Committee, Pulmonary and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD

1999-present **Member**, Internship Selection Committee, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD

1999-present **Member**, Fellowship Selection Committee, Division of Pulmonary and Critical Care Medicine, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD

2001-present **Member,** Fellow Review Committee, Division of Pulmonary and Critical Care Medicine, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD

2003 **Member,** Howard County Comprehensive Health Improvement Plan for the Year 2010, Howard County Health Department, Columbia, MD

2003-present **Member**, Faculty Development Committee, Division of Pulmonary and Critical Care Medicine, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD

2005 **Member**, Curriculum Reform Committee Meeting, Johns Hopkins School of Medicine, Baltimore, MD

2009-present **Member**, Planning Committee, CME Activity – Medical Grand Rounds, Johns Hopkins School of Medicine, Baltimore, MD

2011-present **Director**, Obstructive Lung Disease Program, Division of Pulmonary and Critical Care Medicine

2013 Member, Panel Presentation/Discussion: "writing a successful career development application. Johns Hopkins Professional Development Office, September 25. 2013.

2014 *Ad hoc* Committee for a Department of Biostatistics faculty member's promotion to Associate Scientist.

Professional Societies

Associate, American College of Physicians (ACP)
Fellow, American College of Chest Physicians (ACCP)
Member, American Thoracic Society (ATS)

Member, American Federation for Clinical Research (AFCR)
Member, Central Society for Clinical Research (CSCR)
Member, International Society of Environmental Epidemiology

Committee Memberships

American Academy of Allergy, Asthma and Immunology

2003-Present Member, Genetics and Epidemiology

American Thoracic Society

1999-2006	Course Faculty Member, Education: Methods in epidemiologic, clinical and operations research (MECOR).
2002-Present	Member, Behavioral Science Assembly Long Range Planning Committee
2003-Present	Member, Behavioral Science Assembly Program Committee
2003-2004	Chair Elect, Behavioral Science Assembly Program Committee
2003	Member, IRE/MECOR Planning Retreat Committee
2004-2005	Chair, Behavioral Science Assembly Program Committee
2006-2008	Chair, Behavioral Science Assembly
2006-2008	Member, ATS Board of Directors
2008-2009	Chair, Behavioral Science Assembly Nominating Committee
2008-2010	Member, Environmental and Occupational Heath Assembly, Clinical Research Committee
2008-2010	Member, Environmental and Occupational Heath Assembly Program Committee
2008-2010	Member, Environmental and Occupational Health Assembly Working Group on Epidemiology
2008-2009	Mentor Member, Members in Transition and Training Committee
2009-2011	Member, Grant Review Committee for ATS Foundation-Tobacco-dependence research fund grant.
2010-2015	Member, Drug/Device Discovery and Development Committee
2013-2014	Member, Behavioral Science Assembly Planning Committee
2013-2014	Member, Behavioral Sciences and Health Services Research Assembly Nominating Committee

National Committee for Quality Assurance

2003	Member, COPD Technical Subgroup
2004-Present	Member, Clinical Expert Panel

Pennsylvania Department of Health

2004	Member, Grant Review Committee, Centers of Excellence for Research on Lung Disease Review Panel. Washington, DC.
2010-2011	Member, Pennsylvania Final Performance Review, Master Tobacco Settlement for the Pennsylvania Department of Health, 09-10 Cycle B

State of Maryland

2006-2007 Member, Governor-elect Martin O'Malley's Transition Committee, State of Maryland, Department of Health and Mental Hygiene, December, 2006 to January, 2007.

Clinical Trials & Surveys Corp (C-TASC)

2009-present Member, Institutional Review Board

Qatar National Research Fund

2010-present Reviewer, National Priorities Research Program

Netherlands Asthma Foundation

2012-present Member, Grant Review Section

Conference Organizer, Session Chair (see also Classroom Instruction, pages 19-20**)**

2003 Chair, American Thoracic Society International Conference Session: Assessing Patient Health, Healthcare and Outcomes: Limits of Physician Estimation

Facilitator, American Thoracic Society International Conference Session: Environmental and Genetic Risk Factors for Pediatric Lung Disease.

Chair, American Thoracic Society International Conference Symposium: Impact of Psychosocial Factors on Respiratory Health.

2004 Chair, American Thoracic Society International Conference Symposium: Assessing Asthma Severity and Asthma Control According to National Guidelines: Are our Assessments Working?

Chair, American Thoracic Society International Conference Symposium: Diagnosis and Outcomes in Pediatric Asthma.

Chair, American Thoracic Society International Conference Symposium: Pediatric Asthma.

2005 Chair, American Thoracic Society International Conference Symposium: Health Disparities: Understanding and Addressing Them through Research and Practice.

Chair, American Thoracic Society International Conference Symposium: Implementation of Asthma Severity Measurements in the Real World of Clinical Practice: What Are We Doing Now and What Should Come Next?

2006 Chair, American Thoracic Society International Conference Symposium: The Complex Interaction of Race, Stress and Neighborhood on Respiratory Disease. May 21, 2006.

2007 Chair, American Thoracic Society International Conference Symposium: Current Methods for the Respiratory and Environmental Researcher: A Toolkit for Clinical Investigation

Chair, American Thoracic Society International Conference Symposium: Scientific Writing: How to Publish for Academic Success.

Chair, American Thoracic Society International Conference: Assembly on Behavioral Science Membership Meeting.

2008 Chair, American Thoracic Society International Conference Symposium: Introduction to Data Analysis: Exploring the Great Unknown.

Chair, American Thoracic Society International Conference Symposium: Asthma Severity Versus Asthma Control: What Should We Use in Clinical Practice?

2009 Chair, American Thoracic Society International Conference Symposium: Measuring and Improving the Quality of Care in Lung Disease.

Facilitator, American Thoracic Society International Conference Symposium: Developing Surveys that Measure or Predict.

Facilitator, American Thoracic Society International Conference Symposium: Asthma in the Inner City: A Unique Mix of Allergen and Pollutant Exposures.

2010 Chair, American Thoracic Society International Conference, Poster Session Discussion, New Orleans

Chair, American Thoracic Society International Conference, Scientific Symposium: Individual susceptibility to air pollution.

Chair, American Thoracic Society International Conference, Scientific Symposium: Asthma disparities: Root causes and global solution

2013 Chair, American Thoracic Society International Conference, EOH Program Committee

2013 Chair/Moderator, American Thoracic Society International Conference, Poster Session Discussion, Pollution Effects, Philadelphia

2013 Discussant, American Thoracic Society International Conference, Poster Session Discussion, Obesity: Impact on lung function and disease, Philadelphia

2013 Chair, Scientific Symposium: Developmental origins of asthma and allergies: Environment, modifiers and mediators, American Thoracic Society International Meeting, Philadelphia, May 2013.

2015 Chair, Scientific Symposium: Advances in Understanding and Reducing Asthma Disparities, American Thoracic Society International Meeting, San Diego, May 2015.

Advisory Committees, Review Groups

2001-2006 American Lung Association
Member, National Grants Review Award Selection Committee

2002 American Healthways/Johns Hopkins 2nd Annual Disease Management Outcomes Summit: Standard Outcome Metrics and Evaluation Methodology for Disease Management Programs, November 7-10, 2002, Palm Desert, CA, Role: Physician Steering Committee

2003 American Healthways/Johns Hopkins 3rd Annual Disease Management Outcomes Summit: Defining the Patient-Physician Relationship for the 21st Century, October, 2003, Phoenix, AZ. Role: Physician Steering Committee

Member, Aventis AVE0547 HE Asthma Advisory Board

2004	American Healthways/Johns Hopkins 4th Annual Disease Management Outcomes Summit: Outcomes-Based Compensation: Pay-for-Performance Design Principles, November 11-14, 2004, Rancho Mirage, CA, Role: Physician Steering Committee
	Member, DEY, LP, Managed Care Advisory Board, Napa, CA.
2005	American Healthways/Johns Hopkins 5th Annual Disease Management Outcomes ummit: Improving Care Coordination through Physician-Disease Management Collaboration, November 10-13, 2005, Fort Lauderdale, Florida, Role: Physician Steering Committee
	Invited Faculty representing ATS, National Workshop to Reduce Asthma Disparities, Chicago, Illinois
2006	American Healthways/Johns Hopkins 6th Annual Disease Management Outcomes Summit: Embracing Health: Tools and Systems for Health Promotion and Disease Prevention, November, 2006, JW Marriott Starr Pass Resort, Tucson, AZ , Role: Physician Steering Committee
	Member, NIH/NHLBI Grant Review Award Selection Committee
	Member, NHLBI Strategic Planning Process Committee
2007	American Healthways/Johns Hopkins 7th Annual Disease Management Outcomes Summit: Integrated Medicine: Complementary Approaches, November 8-11, 2007, Austin, Texas, Role: Physician Steering Committee
2008	Reviewer, *ad hoc*, Deuthsche Forschungsgemeinschaft (German Research Foundation).
	Member, Cancer, Cardiovascular and Pulmonary Disease (CCPD) program. The Amendment 25 Program Evaluation Group.
	Member, National Committee for Quality Assurance (NCQA) Advisory Panel. HEDIS Trends Publication Expert Advisory Panel.
2008-	Member, EXPORT's P60 Advisory Board, University of Puerto Rico (UPR)/CHA Research presentCenter of Excellence: Making a Difference for Latino Health. San Juan, Puerto Rico.
2008-2009	Chair, The Donaghue Program for Research Leadership, Hartford, CT.
2009-2011	Member, NIH/NHLBI Study Section for Patient Oriented Research (K23, 24, and 25).
2009	Member, NIH/NIAID Review Panel for Special Emphasis Study Section ZAI1-RRS-I-M1.
2010	Discussant, NIH/NIAID, Asthma, Allergy and Inflammation Branch: Asthma Outcomes Workshop, Bethesda, MD.
2011	Member, NIH/NHLBI Review Panel for Small Business Respiratory Sciences, Special Emphasis Study Section, ZRG1 CVRS-H (10) B (K12)

2011 Member, NIH/NHLBI Review Panel for NHLBI Career Development Programs in Emergency Medicine Research (K12).

2011 Chair, NIH/NHLBI Review Panel for RFA-HL-12-011, Development and testing of a case finding methodology in COPD (R01), Washington, DC

2012 Discussant, Webinar Presentation, NIEHS, Virtual Forum: Childhood Obesity and the Environment, November 2012. Research Triangle Park, NC

Consultancies

Aventis 2002; Physician Advisory Panel

Cardiocontinuum, 1999-2000; Role: Development of COPD Program

American Healthways, 2002-Present; Role: Steering Committee Member and Performance Measure Development

Sorption Technologies, Inc., 2004-Present; Role: Research Design Consultant

Interactive Forums, Inc., 2004-Present; Role: Health Care Consulting

Merck, Beta-agonist Measure Panel Meeting, December 3, 2004, Denver, Colorado.

Pfizer Academic Round Table, May 24-25, 2005, American Thoracic Society, San Diego, California.

RECOGNITION

Awards, honors

1986 English Degree awarded with Honors, University of Pennsylvania

1986 BA, *Magna cum Laude*, University of Pennsylvania

1986 BS, *Magna cum Laude*, University of Pennsylvania

1997 Delta Omega Public Health Honor Society

2000 Solo Cup Clinician Scientist Award

2001 GlaxoSmithKline Development Partners' Junior Faculty Award

2009 Qforma's List of Most Influential Doctors, created for USA Today.

2010 Pfizer Visiting Professorship in Pulmonology.
East Tennessee State University College of Public Health.

Invited Talks, Panels

1993 The Special Value of Undergraduate Research. Presented at the 64th Annual Meeting of the Eastern Psychological Association. Arlington, Virginia.

PSA as a Screening Test? Medical Management Conference. Department of Internal Medicine, University of Pennsylvania.

1994 Carbon Monoxide Poisoning, Medical Management Conference, Department of Internal Medicine, University of Pennsylvania.

Invited Discussant, Morbidity and Mortality Conference, Department of Internal Medicine, University of Pennsylvania.

1996 Vitamins and the Risk of Lung Cancer: Randomized Clinical Trials as a Gold-Standard, Longcope Attending Rounds, Department of Internal Medicine, The Johns Hopkins University School of Medicine.

PSA and DRE Screening for Prostate Cancer: Principles of Screening, Longcope Attending Rounds, Department of Internal Medicine, Johns Hopkins University School of Medicine.

1997 Predictors of overuse of inhaled β-agonists, underuse of inhaled corticosteroids, and of nocturnal symptoms in adult asthmatics. Outcomes research group, Merck & Co., Inc., West Point, PA.

Associations of misuse of asthma medications in adult asthmatics enrolled in managed care. Managed Care Health Care Consortium, Washington, DC.

Misuse of corticosteroid and β-agonist metered dose inhalers (MDIs) among adult asthmatics in managed care (MCOs), Maryland Thoracic Society Annual Research Dinner, Baltimore, MD.

1998 Treatment patterns among adult asthmatics: Overuse of inhaled beta-agonists, underuse of inhaled corticosteroids, Division of Pulmonary and Critical Care Medicine, Yale University School of Medicine, New Haven, CT.

Treatment patterns among adult asthmatics: Overuse of inhaled beta-agonists, underuse of inhaled corticosteroids, Division of General Internal Medicine, Case Western University School of Medicine, Cleveland, OH.

Misuse of corticosteroid and β-agonist metered dose inhalers (MDIs) among adult asthmatics in managed care (MCOs), Combined Allergy and Immunology Meeting, Palm Beach, FL.

Future HEDIS Measures for Asthma. Glaxo-Wellcome Asthma Managed Care Consultants Program. Naples, FL.

Asthma Therapy Assessment Questionnaire: Results of a Validation Study, Blue Plus, Minneapolis, MN.

1999 Bronchoscopy Quality Improvement Project: A Hospital Based Cohort Study. Health Services Research and Development Research Seminar.

Lesson Learned from Studies of Asthma in Managed Care. Best Practices Symposium sponsored by the Pacific Business Group on Health, Oakland, California.

Quality of Care and Guidelines: Management of Asthma. Practice Guidelines Workshop. Johns Hopkins Medical Services Corporation, Baltimore, MD, November 1999 and May 2000.

2000 Asthma Care by Asthma Specialists. Department of Medicine Grand Rounds. Greater Baltimore Medical Center, Baltimore, MD.

Predictors of Outcomes in Asthma. Frontiers in Research and Clinical Management of Asthma and Allergy Conference. Johns Hopkins Asthma & Allergy Center, Baltimore, MD.

Update in Asthma. Update in Pulmonary and Critical Care Medicine, Johns Hopkins University, Santa Fe, NM.

Fine-tuning your Bronchoscopy Practice. Bronchoscopy Workshop. Johns Hopkins University, Santa Fe, NM.

Underuse of Inhaled Corticosteroids in Asthma. Department of Medicine Grand Rounds, Johns Hopkins University, Baltimore, MD.

Nocturnal Asthma: Impact on Children and Their Parents. Research Conference of the Center for Childhood Asthma in the Urban Environment, Johns Hopkins University, Baltimore, MD.

Bronchoscopy Quality Improvement Project: Design Issues and Results. Robert Wood Johnson Clinical Scholars Program, Johns Hopkins University, February 1998 and April 2000.

2001 COPD- The Role of Steroids. Maryland Thoracic Society 41st Annual Meeting and Scientific Session, Pulmonary and Critical Care Medicine: State-of-the-Art, Baltimore, MD.

Update in Asthma. Johns Hopkins Bayview Medical Center, Department of Medicine, Baltimore, MD.

Severity, Control and Nocturnal Symptoms of Asthma in Children. Research Conference, Division of Pediatric Pulmonary Medicine, Johns Hopkins University. Baltimore, MD.

2002 Non-pharmacologic pain control with Bedscapes for Bronchoscopy. American Red Cross, Arlington, VA.

2003 Annual High Sierra Critical Care Conference; Update in Asthma Management for 2003.

Annual High Sierra Critical Care Conference; How to get the most from your bronchoscopy practice.

Office of Community Health, Community Chats 2002-2003.

Burnt Pizza and Near-Death from Asthma. Department of Internal Medicine Grand Rounds, Johns Hopkins Bayview Medical Center, Baltimore, Maryland.

Using Functional Genomics to Understand Complex Lung Disease, ATS/NHLBI.

Aligning Asthma Care with Assessment of Severity, Healthcare Quality and Safety Research Seminar Series, JHU.

Asthma Epidemiology, World Allergy Organization (WAO), Vancouver.

Aligning Asthma Care with Assessment of Severity. Pulmonary and Critical Care Grand Rounds, Oregon Health Services University.

2004 Office of Community Health, Community Chats 2003-2004; Impact of Night Time Asthma on Children and their Families Effective Asthma Medication.

The Role of the Indoor Home Environment in Childhood Asthma. Johns Hopkins-Barbados Genetic Epidemiology of Obstructive Lung Disease Research Conference, Almond Bay, Hastings, Christ Church, Barbados.

Environmental Factors Impacting Respiratory and Immunologic Disease. Gulf Coast Pediatric Environmental Health Symposium, Baylor College of Medicine, Houston, Texas.

Aligning Asthma Care with Estimates of Asthma Severity: Development of the Asthma Communication Instrument. Research Conference, Division of Pulmonary and Critical Care Medicine, Department of Pediatrics, Johns Hopkins University, Baltimore, Maryland.

Epidemiology as a Tool for Understanding Respiratory Disease: Case-Control Studies, American Thoracic Society, Orlando, Florida.

Standardizing the Care of the Patient with COPD: Is the Quality of Care Truly Improved? American Thoracic Society, Orlando, Florida.

Getting the Most Out of Bronchoscopy Services, 6th Annual Update, Pulmonary and Critical Care Medicine, Santa Rosa, California.

Severe Asthma: Current and Future Management, 6th Annual Update, Pulmonary and Critical Care Medicine, Santa Rosa, California.

2005 Office of Community Health, Community Chats 2005-2006. The Growing Child and Other Health Issues: Impact of Night-Time Asthma on Children and Their Families

Office of Community Health, Community Chats 2005-2006; Lung Disease: Making the Home Safer for Asthmatics.

Health Care Communications and Cultural Competency, National Workshop to Reduce Asthma Disparities, Chicago, Illinois.

The Home Environment of East Baltimore Preschool Children With and Without Asthma, Department of Physiology, Bloomberg School of Public Health, Johns Hopkins University.

COPD: A Pragmatic Approach to Improving Outcomes. Baltimore, Maryland.

COPD: A Pragmatic Approach to Improving Outcomes. COPD Exchange, Pittsburgh, Pennsylvania.

COPD: Evolving Concepts of Therapy. COPD Exchange, Baltimore, Maryland.

Aligning Asthma Care with Assessment of Severity and Control in Practice. Department of Internal Medicine, York Hospital, York, Pennsylvania.

Office of Community Health, Community Chats 2005-2006; Treating Asthma in Children, New Psalmist Christian School, Baltimore, Maryland.

The Role of the Indoor Home Environment in Childhood Asthma. Johns Hopkins-Barbados Asthma Conference, Almond Bay, Hastings, Christ Church, Barbados.

Is it Smart to Prescribe Long-Acting β-Agonists for Patients with Asthma? Division of Allergy and Clinical Immunology, Johns Hopkins University. December 2, 2005; and Rush University, Chicago, Illinois.

Aligning Asthma Care with Assessment of Severity and Control in Practice. Primary Care Conference, Baltimore, Maryland, February 24, 2006;
Ohio State Pulmonary Grand Rounds, April 7, 2006; and
Hospital of the University of Pennsylvania. January 25, 2008.

2006 Aligning Asthma Care with Assessment of Severity and Control in Practice. American Lung Association, Chicago, Illinois.

Should We Still use Long-acting Beta-Agonists in Patients with Asthma? Johns Hopkins University, School of Medicine, Department of Medicine Grand Rounds.

Development of the Asthma and Control Communication Instrument. University of Maryland, Pulmonary Research Conference, Baltimore, MD.

Update in COPD. Baltimore-Washington Hospital, Department of Medicine Grand Rounds. Glen Burnie, Maryland.

Issues Related to Beta-2 Agonist Therapy; Polymorphisms/Clinical Outcomes/Adverse Events Profile. 20th Annual Update. Frontiers in Research and Clinical Management of Asthma and Allergy: From Bench to Bedside. Johns Hopkins University School of Medicine, Division of Allergy and Clinical Immunology, Johns Hopkins Asthma & Allergy Center at Johns Hopkins Bayview Medical Center, Baltimore, Maryland.

2007 Office of Community Health, Community Chats 2007-2008; Asthma: How Asthmatics Can Make Their Home Safer; Effective Asthma Medication.

Hyperinflation in COPD Linking Physiology to Patient Experience. Boehringer-Ingelheim Pharmaceuticals, Inc, Christiana Care Hospital, Newark, Delaware.

Environmental Issues in Managing Asthma. 41st Respiratory Care Journal Conference. Scottsdale, Arizona, September 28, 2007.

Translational Science Think Tank. Collaborative Research Bridging Basic, Clinical and Health Services Domains: Challenges and Opportunities." University of Connecticut Health Center, Farmington, CT, December 6, 2007.

2008 NCQA On-line Program: Best Practices in COPD Treatment. Course Faculty. December 2007-December 2008.

Approaching and Garnering the Support of Community Partners for Community-Based Research. American Thoracic Society International Meeting.

Logistic Regression. American Thoracic Society International Meeting Post-graduate Course.

Assessing Control is Good, But Not Sufficient for Management of Asthma. American Thoracic Society International Meeting Scientific Symposium.

The Death of Primary Care. Barker Grand Rounds, Johns Hopkins University, Baltimore, Maryland.

Is Genetic Polymorphism important in response to asthma therapy? Johns Hopkins 21st Update Frontiers in Research and Clinical Management of Asthma and Allergy. Johns Hopkins Asthma and Allergy Center, Baltimore, Maryland.

Joint Indo-US Workshop on Environmental Risks of Respiratory Disease. Prevalence of Respiratory Disease in India. Chandigarh, India.

Bridging the Evidence-to-Practice Gap in Asthma and Chronic Obstructive Pulmonary Disease from a National and International Perspective: An Update. American Thoracic Society International Meeting, San Diego, CA.

2009 Diet and inner city asthma: Is there a connection? Department of Medicine Grand Rounds, Johns Hopkins University, Baltimore, MD.

Role of indoor pollutants in respiratory disease. Fellows Orientation Conference, Division of Allergy and Clinical Immunology, Johns Hopkins University, Baltimore, MD.

Susceptibility determinants of childhood asthma. Session: Contributing factors that influence the relationship between environmental exposures and children's health. Pediatric Academic Societies Annual Meeting, Baltimore, MD.

Scientific Advisory Committee, Merck Childhood Asthma Network, Washington, DC.

Pediatric Asthma Roundtable meeting-Improve lives of children with asthma in the Baltimore area. National Asthma Campaign, Baltimore, MD.

2010 Topics in Clinical Medicine 2010. Session: Meet the Professor—Pulmonary. Johns Hopkins University Annual Topics in Clinical Medicine, Baltimore, MD.

Validated questionnaires in the management of allergic disorders: Applications and interpretation. Johns Hopkins Community Physicians, Baltimore, MD.

Validated questionnaires in the management of allergic disorders: Effective Use in an Allergy Practice Setting. Session: State-of-the-Art Session 2525: American Academy of Allergy, Asthma and Immunology International Meeting, New Orleans, LA.

Indoor environmental exposures and asthma disparities. Scientific Symposium: Asthma disparities: Root cause and global solution. American Thoracic Society International Meeting, New Orleans, LA.

2010 Speaker: Environmental Issues in Managing Asthma. Post-Graduate Respiratory Medicine Meeting, Irish Lung Foundation, Dublin, Ireland. June 2010.

Speaker, Asthma Update Seminar. Eastern Shore AHEC, Hyatt Regency Chesapeake Bay, Cambridge, Maryland. August 2010.

Academy of Industrial Hygiene, PCIH 2010. Fort Worth, Texas, October 7-8, 2010
21st Century Toxicity Testing and Human Health Risk Assessment for Environmental Agents.
Speaker: (1) "Lung responses to environmental toxins"
Speaker: (2) "Environmental residential exposures to allergens and irritant gases"
Speaker: (3) "Role of Pulmonary and Respiratory Irritants in Asthma, COPD, and Bronchiolitis Obliterans"

Speaker: "Is diet driving the asthma epidemic?" NIEHS/EPA Conference, Protecting children's health for a lifetime: Environmental health research meets clinical practice and public policy conference, October 19-20, Washington, DC

Speaker: Environmental Health Department Doctoral Seminar, Boston University, October 22, 2010. "The role of indoor pollutants and allergens and asthma in inner city children: Some of the bad ingredients in a toxic stew."

Participant, Workshop: Task Force for the Asthma Disparities Working Group/Federal Task Force on Environmental Health Risks and Safety Risks to Children Steering Committee, "Developing a coordinated federal action plan to reduce asthma disparities." NIH-NHLBI/EPA/HUD. Washington, DC. December 16-17, 2010.

2011 Visiting Professor, Leading Voices in Public Health Lecture Series. "The mouse, the house and the hamburger: Making sense of the asthma epidemic." The College of Public Health and the Public Health Student Association, East Tennessee State University, March 3, 2011.

Lecturer, Teaching Course entitled Health Care Organization and Delivery: "Indoor environmental exposures and asthma disparities." East Tennessee State University, March 3, 2011.

Lecturer, Teaching Course entitled Introduction to Air Pollution: "Asthma and Air Pollution." East Tennessee State University, March 4, 2011.

Invited Speaker: U.S. Congress Briefing, Preventing Breast Cancer and Pediatric Asthma: Links to the Environments of Women and Children, Rayburn House Office Building B-354 NIH/NIEHS. "The Mouse, the House and the Hamburger: Making Sense of the Asthma Epidemic." April 21, 2011.

Invited Speaker-Panelist: Clearing the Air, Addressing asthma disparities in Maryland.
Session A-3: "Asthma Interventions: Research into Practice," and
Session B-4: "The human side of asthma: Educating patients to make health decisions—overcoming barriers to medication adherence." Linthicum, MD. June 2011.

Invited Speaker: National Healthy Homes Conference. Track 7: Just the Facts. Session 7H-2. "Nanoparticles and nitrogen dioxide from stoves: Health effects and strategies to reduce exposure and improve asthma control." Denver, CO. June 2011.

2012 Visiting Professor, Division of Pulmonary Medicine, Allergy and Immunology Children's Hospital of Pittsburgh of UPMC, Pittsburgh, PA, January 5, 2012.

Invited Speaker, Pediatric Pharmacology Division, National Jewish Health. "The house, the mouse and the hamburger: Making sense of the asthma epidemic." Denver, CO, June 2012.

Invited Speaker, Johns Hopkins Bloomberg School of Public Health/The Maryland Department of Health and Mental Hygiene/The mid-Atlantic Public Health Training Center. "Reducing asthma disparities in children: A model program with promising results. Baltimore, MD, June 2012.

Invited Speaker, EPA/NIEHS Children's Centers 2012 Webinar Series, Protecting children's health for a lifetime. "Role of home environment and diet on childhood asthma." December 2012.

2013 Visiting Professor, "The house, the mouse and pizza: Explaining the asthma epidemic." Universidad de Los Andes, Bogota, Columbia, April 2013.

Invited Speaker, Scientific Symposium: Developmental origins of asthma and allergies: Environment, modifiers and mediators, "Indoor exposures and ETS." American Thoracic Society International Meeting, Philadelphia, PA. May 2013.

Invited Speaker, Congressional Briefing, Health and Medicine Counsel of Washington. "Protecting children's health for a lifetime: How the environment influences health and development," hosted by Senator Kirsten E. Gillibrand, 385 Russell Senate Office Building. Sponsored by Friends of NIEHS, the American Academy of Pediatrics, and the Children's Environmental Health Network. October 2013.

Invited Lecturer, Johns Hopkins University School of Nursing, "Diagnosis, Symptom, and Illness Management I – Adult Course." Topic: Asthma. December 2013.

2014 Invited Lecturer and participant, NIH – MOST Clinical and Translational Science Workshop, NIH Campus, Stone House, Bethesda, MD, July 21-22, 2014.

Invited Speaker, Respiratory Expert Forum Ireland, "Beat the Professor" Case Studies on treating difficult airways disease, Dublin, Ireland, October 17-18, 2014.

2015 Invited Speaker, The Children's Environmental Health Network's 2015 CEHN Pediatric Research Conference Children: Food and Environment, "Prevention and Treatment of Asthma with Diet: Progress and Promise." The University of Texas at Austin, Austin, TX. February 4-6, 2015.

Invited Speaker, Scientific Symposium: Advances in Understanding and Reducing Asthma Disparities, "Indoor Exposures and Asthma Disparities." American Thoracic Society International Meeting, San Diego, CA. May 2015.

Office of Community Health, Community Chats 2015-2016; Asthma: "How People With Asthma Can Make Their Homes Safer."

Office of Community Health, Community Chats 2015-2016; Asthma: "Does Diet Affect Asthma?"

APPENDIX D

Gregory Diette, MD, MHS

Diette publications, continued, after June 2017

1. Brigham EP, Steffen LM, London SJ, Boyce D, **Diette GB**, Hansel NN, Rice J, McCormack MC. Diet Pattern and Respiratory Morbidity in the Atherosclerosis Risk in Communities Study. *Annals of the American Thoracic Society.* 2018; 15(6).

2. Brigham EP, Matsui EC, Appel LJ, Bull DA, Curtin-Brosnan J, Zhai S, White K, Charleston JB, Hansel NN, **Diette GB**, McCormack MC. A pilot feeding study for adults with asthma: The healthy eating better breathing trial. *PLOS ONE.* 2017; 12(7).

3. Cloutier MM, Salo PM, Akinbami LJ, Cohn RD, Wilkerson JC, **Diette GB**, Williams S, Elward KS, Mazurek JM, Spinner JR, Mitchell TA, Zeldin DC. Clinician Agreement, Self-Efficacy, and Adherence with the Guidelines for the Diagnosis and Management of Asthma. *The Journal of Allergy and Clinical Immunology: In Practice.* 2018; 6(3): 886-894.

4. Lin SY, Azar A, Suarez -Cuervo C, **Diette GB**, Brigham E, Rice J, Ramanathan M, Gayleard J, Robinson KA. The Role of Immunotherapy in the Treatment of Asthma. *AHRQ Comparative Effectiveness Reviews, No. 196.* 2018.

5. Lin SY, Azar A, Suarez-Cuervo C, **Diette GB**, Brigham E, Rice J, Ramanathan Jr. M, Robinson KA. Role of sublingual immunotherapy in the treatment of asthma: An updated systematic review. *International Forum of Allergy and Rhinology.* 2018; 8(9): 982-992.

6. McCormack MC, Paulin LM, Gummerson CE, Peng RD, **Diette GB**, Hansel NN. Colder temperature is associated with increased COPD morbidity. *European Respiratory Journal.* 2017; 49(6).

7. Nnodum BN, McCormack MC, Putcha N, Hwang S, Paulin LM, Brigham EP, Fawzy A, Romero K, **Diette GB**, Hansel NN. Impact of Physical Activity on Reporting of Childhood Asthma Symptoms. *Lung.* 2017; 195(6): 693-698.

8. Paulin LM, Williams DL, Peng R, **Diette GB**, McCormack MC, Breysse P, Hansel NN. 24-h Nitrogen dioxide concentration is associated with cooking behaviors and an increase in rescue medication use in children with asthma. *Environmental Research.* 2017; 159: 118-213.

9. Rice JL, **Diette GB**, Suarez-Cuervo C, Brigham EP, Lin SY, Ramanathan Jr. M, Robinson KA, Azar A. Allergen-Specific Immunotherapy in the Treatment of Pediatric Asthma: A Systematic Review. *Pediatrics.* 2018; 141(5).

10. Rice JL, Brigham E, Dineen R, Muqueeth S, O'Keefe G, Regenold S, Koehler K, Rule A, McCormack M, Hansel NN, **Diette GB**. The feasibility of an air purifier and secondhand smoke education intervention in homes of inner city pregnant women and infants living with a smoker. *Environmental Research. 2018;* 160: 524-530.

11. Sulaiman I, Greene G, MacHale E, Seheult J, Mokoka M, D'Arcy S, Taylor T, Murphy DM, Hunt E, Lane SJ, **Diette GB**, FitzGerald JM, Boland F, Bhreathnach AS, Cushen B, Reilly RB, Doyle F, Costello RW. A randomized clinical trial of feedback on inhaler adherence and technique in patients with severe uncontrolled asthma. *European Respiratory Journal.* 2018; 51(1).

12. Wu TD, Eakin MN, Rand CS, Brigham EP, **Diette GB**, Hansel NN, McCormack MC. In-Home Secondhand Smoke Exposure Among Urban Children With Asthma: Contrasting Households With and Without Residential Smokers. *Journal of Public Health Management and Practice.* 2018; 25(2): E7-E16.

13. Wu TD, Brigham EP, Peng R, Koehler K, Rand C, Matsui EC, **Diette GB**, Hansel NN, McCormack MC. Overweight/obesity enhances associations between secondhand smoke exposure and asthma morbidity in children. *The Journal of Allergy and Clinical Immunology: In Practice.* 2018; 6(6): 2157-2159.

14. POSTER DISCUSSION SESSION:

    Nnodum BN, Hwang S, Romero K, Kineza C, Tariq Z, Peng R, Putcha N, McCormack MC, Diette GB, Hansel NN. Impact Of Physical Activity On Childhood Asthma Symptoms: Longitudinal Study In Inner City Baltimore, Maryland. *Poster Discussion Session/ Wednesday May 24 2017/ Walter E. Washington Convention Center*

15. POSTER DISCUSSION SESSION:

    Wu TD, Eakin M, Rand CS, Brigham E, Diette GB, Hansel NN, McCormack MC. Factors Associated with In-Home Secondhand Smoke Exposure from External Sources in Urban Children with Asthma. *Poster Discussion Session/ Sunday May 20/ San Diego Convention Center*

16. POSTER DISCUSSION SESSION:

    Wu TD, Brigham E, Rand CS, **Diette GB**, Peng R, Putcha N, Koehler K, Hansel NN, McCormack MC. Overweight and Obesity Increases Respiratory Symptoms Associated With Secondhand Smoke Exposure Among Us Children. *Poster Discussion Session/ Wednesday May 24/ Walter E. Washington Convention Center*

17. POSTER DISCUSSION SESSION:

    Koch A, Woo H, Brown RH, Brooker A, Paulin LM, Schneider H, Schwartz AR, Diette GB, Wise RA, Hansel NN, Putcha N. Obstructive Sleep Apnea is Associated with Airway Dimensions in COPD. *Poster Discussion Session/ Tuesday May 22, 2018/ Marriott Marquis San Diego Marina*

18. POSTER DISCUSSION SESSION:

    Liesching TN, Huynh T, Cereda M, **Diette GB**. Treatment with the MetaNeb® System in High-Risk Post-Surgical Patients Reduced Hospital and Intensive Care Unit Length of Stay. *Poster Discussion Session/Sunday May 20/San Diego Convention Center*

19. POSTER DISCUSSION SESSION:

    Polito C, Eakin M, Woo H, Romero K, McCormack MC, Fawzy A, Paulin LM, **Diette GB**, Koehler K, Hansel NN, Putcha N. Indoor Air Pollution May Be Associated with cognitive Impairment in Chronic Obstructive Pulmonary Disease. *Thematic Discussion Session/Monday May 21/San Diego Convention Center*

20. POSTER DISCUSSION SESSION:

Putcha N, Fawzy A, Matsui E, Bowler RP, Woodruff P, O'Neal WK, Comellas AP, Han MK, Dransfield MT, Lugogo N, Hoffman EA, Cooper CB, Hersh CP, Paulin LM, Drummond M, Wise RA, **Diette GB**, Hansel NN. Allergen Sensitization and Exposure Is Associated with Exacerbations in COPD. *Poster Discussion Session/Monday May 21/San Diego Convention Center*

21. POSTER DISCUSSION SESSION

    Rice J, Brigham EP, Koehler K, McCormack MC, **Diette GB**, Woo H, Hanson C, Sharma S, Kolahdooz F, Hansel NN. Adherence to a Mediterranean Diet Attenuates the Adverse Effect of Indoor Particulate Matter on Asthma Symptoms in Children. *Poster Discussion Session/ Tuesday May 22/ San Diego Convention Center*

22. THEMATIC POSTER SESSION:

    Wu TD, Rice J, Koehl R, Brigham E, **Diette GB**, Hansel NN, Sterni LM, McCormack MC. Pediatric Sleep Disordered Breathing is Associated with Worse Acute Asthma Control. *Thematic Poster Session/ Sunday May 20, 2018/ San Diego Convention Center*

23. THEMATIC POSTER SESSION:

    Cereda M, Huynh T, Liesching T, **Diette GB**. Identification Of Surgical Population At High Risk Of Postoperative Pulmonary Complications. *Thematic Poster Session/ Sunday May 21, 2017/ Walter E, Washington Convention Center*

24. THEMATIC POSTER SESSION:

    Soto, CML, Woo H, Romero K, Brigham E, McCormack MC, **Diette GB**, Hanson C, Fawzy A, Koch A, Putcha N, Hansel NN. Association of Omega-3 and Omega-6 Fatty Acid Intake with Inflammation and Respiratory Outcomes in COPD. *Thematic Poster Session/ Monday May 21, 2018/ San Diego Convention Center*

25. MINI SYMPOSIUM:

    Bose S, McCormack MC, Woo HS, Romero K, Brigham E, Koehler K, Detrick B, **Diette GB**, Hansel NN. Vitamin D Status Modifies Response to Indoor Air Pollution in Urban Children with Asthma. *Mini Symposium/ Sunday May 20/ San Diego Convention Center*

26. MINI SYMPOSIUM:

    Brigham E, McCormack MC, Woo H, Rice J, Koehler K, Vulcain T, Wu TD, Biswal SS, Sudini K, Koch A, Hanson C, Sangita S, Kolahdooz F, Bose S, Romero K, **Diette GB**, Hansel NN. Omega-3 and Omega-6 Fatty Acid Intake Modifies Response to Indoor Air Pollution in Children with Asthma. *Mini Symposium/ Sunday May 20/ San Diego Convention Center.*

27. Listed as a Reviewer and Technical Contributor:

    World Health Organization. Air Pollution and Child Health: Prescribing Clean Air Summary. 2018.

APPENDIX E

Date	Case Name	Case Number	Deposition or Trial
16-Jan-2014	Ismael Rosas v. Flavorchem Corporation, et al	Superior Court of the State of California Count of Los Angeles, Central Civil West Case No.: BC400974	Deposition (O'Laughlin Industries)
1-Aug-2014	Tanu Vatuvei v. Mission Flavors & Fragrances, Inc., et al.	Superior Court of the State of California for the county of Orange, Central Justice Center Case No.: 30-2011-00518123	Deposition (O'Laughlin Industries)
10-Jun-2014	Harry Goldsmith v. ACandS, Inc., et al. (Law Offices of Peter G. Angelos: Mr. William Minkin, Esq.)	In the Circuit Court for Baltimore City Case No.: 24x13000097	Deposition (Hampshire Industries)
3-Oct-2014	Charles Waters v. ACandS, Inc., et al. (Law Offices of Peter G. Angelos: Mr. Gary Ignatowsi, Esq.)	In the Circuit Court for Baltimore City Case No.: 24x13000461	Deposition (Hampshire Industries)
31-Oct-2014	Francis Murphy v. ACandS, Inc., et al. (Law Offices of Peter G. Angelos: Mr. William Minkin, Esq.)	In the Circuit Court for Baltimore City Case No.: 24x13000371	Deposition (Hampshire Industries)
19-Feb-2015	Rachele and David Ventres v. 002 Auto Parts Inc., et al. (Levy Konigsberg: Joseph Mandia, Esq.)	Superior Court of New Jersey Case No.: MID-L-1933-12AS	Deposition (BASF)
19-Feb-2015	Thomas and Donna Gioglio v. 3M Company, et al. (Levy Konigsberg: Joseph Mandia, Esq.)	Superior Court of New Jersey Case No.: MID-L-4593-12AS	Deposition (BASF)
25-Feb-2015	Lorene McKenzie, deceased v. Palestine Principal Healthcare Limited Partnership, et al.	District Court of Anderson County Texas, 369th Judicial District. Case No.: 369-12-4684	Trial (Plaintiff)
13-Mar-2015	Walter Henry Hakenjos v. AT&T Corporation, et al. (Cannella Law Firm: David Cannella, Esq.)	Civil District Court for the Parish of Orleans State of Louisiana Case No.: 14-3828	Deposition (AT&T)
10-Apr-2015	Robert Menoche (as part of Raymond Michaels, et al.,) v. ACandS, Inc., et al. (Law Offices of Peter G. Angelos: Mr. Theodore Fierlage, Jr., Esq.)	In the Circuit Court for Baltimore City Case No.: 24x14000259	Deposition (Hampshire Industries)

Date	Case Name	Case Number	Deposition or Trial
22-May-2015	Kathy Mason v. Vistas at Lake Largo, LLC (Eccleston and Wolf: Mark Johnson, Esq.)		Deposition (de bene esse) (Vistas at Lake Largo)
3-Jun-2015	Senate Committee on Environment & Public Works	Challenges and Implications of EPA's Proposed National Ambient Air Quality Standard for Ground-Level Ozone and Legislative Hearing on S. 638, S. 751, and S. 640.	(Minority)
12-Jun-2015	Donald Russ and Ann Russ v. Alcatel-Lucent USA Inc, et al. (Simmons Hanley Conroy: Daniel Blouin, Esq.)	In the Superior Court of New Jersey Case No.: MID-L-1249-14-AS	Deposition (AT&T)
16-Jun-2015	House Committee on Energy & Commerce	EPA's Proposed Ozone Rule: Potential Impacts on Manufacturing.	(Minority)
31-Jul-2015	Eric Heggie, as Special Administrator of the Estate of Karry Heggie, Deceased v. Honeywell International, Inc., Et al. (Wylder Corwin Kelly LLP)	In the Circuit Court of the Eleventh Judicial Circuit County of McLean Case No.: 12 L 87	Deposition (Lincoln Electric Company; Hobart Brothers Company)
23-Oct-2015	Kris Penny v. AT&T Corporation, et al. (The Ruckdeschel Law Firm, LLC)	In the United States District Court Middle District of Florida Orlando Division Case No.: 6:15-cv-557-ORL-31KRS	Deposition (AT&T)
25-Aug-2016	Wanda Allen, Individually and as Personal Representative of the Estate of Byron K. Allen, et al. (Dumer & Barnes, P.A.)	In the Circuit Court for Baltimore City Case No.: 24-C-15-003256 OT	Deposition (Clinical Associates, PA: Sinai Hospital of Baltimore, Inc.)
28-Sep-2016	Rudiger & Joan Herion v. Donley's Inc., et al. (Bevan & Associates LPA, Inc.)	In the Court of Common Pleas, Cuyahoga County, Ohio Case No.: 15 CV 848879	Deposition (Donley's Inc.)
11-Jan-2017	Anita M. Albright v. Kevin Anthony Seymour (Law Office of Neil J. Bixler, P.A.: Neil Bixler, Esq.)	Carroll County Circuit Court in Maryland	Trial (Kevin Anthony Seymour)

Date	Case Name	Case Number	Deposition or Trial
3-Feb-2017	Brian Tucker and Sherri Tucker, his wife v. Momentive Performance Materials USA, Inc., et al. (Motley Rice, LLC: Scott B. Hall, Esq.)	In the United States District Court for the Southern District of West Virginia at Charleston Civil Action No. 2:13-cv-04480	Deposition (Joint Defense)
3-Mar-2017	Dennis John Zampa and Pamela S. Zampa v. Georgia-Pacific LLC, et al. (Kazan, McClain, Satterley & Greenwood: Trey Jones, Esq.)	In the Alameda County Superior Court of California Case No.: RG16836998	Deposition (E.I. Du Pont de Nemours and Company)
8-Mar-2017	Gregory Aregood, Jr., et al. v. International Flavors & Fragrances, Inc., et al. (Humphrey, Farrington & McClain, P.C.: Steven E. Crick, Esq.)	United States District Court for the Southern District of Indiana Civil Action No.: 1:14-CV-00274-LRM-TAB	Deposition (Givaudan Flavors Corporation)
24-Mar-2017	Gregory Aregood, Jr., et al. v. International Flavors & Fragrances, Inc., et al. (Humphrey, Farrington & McClain, P.C.: Steven E. Crick, Esq.)	United States District Court for the Southern District of Indiana Civil Action No.: 1:14-CV-00274-LRM-TAB	Continued Deposition (Givaudan Flavors Corporation)
29-Mar-2017	Dennis John Zampa and Pamela S. Zampa v. Georgia-Pacific LLC, et al. (Kazan, McClain, Satterley & Greenwood: Trey Jones, Esq.)	In the Alameda County Superior Court of California Case No.: RG16836998	Deposition (E.I. Du Pont de Nemours and Company)
6-Sep-2017	Aaron Ruby, et al., v. International Flavor & Fragrances, INC., et al. (Stephen J. Butler, Esq.)	Court of Common Pleas Marion County, Ohio Case No.: 2014 CV 0509	Deposition (Givaudan Flavors Corporation)
14-Dec-2017	Terry Darpel, et al. v. Cargill Flavor Systems US, LLC, et al. (Motley Rice, LLC: Scott B. Hall, Esq.)	Commonwealth of Kentucky Kenton Circuit Court, Division III. Case No.: 12-CI-446	Deposition (Emoral; Berje Incorporated)
17-Jan-2018	Delbert Cohen, Individually, and as Personal Representative of the Estate of Muriel Cohen, et al., v. 84 Lumber Company, et al. (The Ruckdeschel Law Firm, LLC; Z. Stephen Horvat, Esq.)	In the Circuit Court for Prince George's County Case No.: CAL16-37427	Deposition (Hampshire Industries)

Date	Case Name	Case Number	Deposition or Trial
4-Apr-2018	Darrell Palmer and Norma Palmer v. Appleton GRP, LLC d/b/a Appleton Group and Emerson Electric Co., et al. (Geoge & Farinas, LLP)	In the Marion Superior Court SS: Civil Division Room 2 Cause No. 49D02-1704-MI-016728	Deposition (Rockwell Automation; Reliance Electric)
22-Apr-2018	Gail Lucille Ingham and Robert Ingham, et al. v. Johnson & Johnson; Johnson & Johnson Consumer Companies, Inc.; and Imerys Talc America, Inc., f/k/a Luzenac America, Inc. (The Lanier Law Firm; Sam E. Taylor, Esq.)	In the Circuit Court of the City of St. Louis State of Missouri Cause No. 1522-CC10417-01	Deposition (Johnson & Johnson)
10-Jul-2018	Blades, Kevin, et al. v. Emoral, Inc., f/k/a Polarome International, Inc., et al. (Humphrey, Farrington & McClain; Scott A. Britton-Mehlisch)	In the Circuit Court of Jasper County, Missouri Case No. 17AO-CC00025	Deposition (Emoral)
27-Jul-2018	Herman Leischner and Bonnie Leischner v. Aerco International, Inc., et al. (Wylder Corwin Kelly LLP; Stephen Wood, Esq.)	In the Circuit Court of the Eleventh Judicial Circuit County of McLean No. 15 L 53	Deposition (Hobart Brothers and Lincoln Electric)
3-Aug-2018	Marlin Herbst v. Bush Boake Allen, Inc., et al. (Humphrey, Farrington & McClain; Michael S. Kilgore, Esq.)	In the United States District Court Northern District of Iowa Western Division No. C17-4008-MWB	Deposition (Givaudan Flavors Corporation & Emoral, Inc.)
30-Aug-2018	Rosalind Henry and Frederick C. Henry v. Brenntag North America, et al. (Motley Rice LLC; W. Christopher Swett, Esq.)	Superior Court of New Jersey, Middlesex County No. MID-L-1748-17AS	Deposition (Johnson & Johnson, Johnson & Johnson Consumer Inc., and Imerys Talc America, Inc.)
28-Sep-2018	Nelcome Courville, Jr. v. Lamorak Insurance Company, et al. (Roussel & Clement; Gerolyn P. Roussel, Esq.)	Civil Dirstric Court for the Parish of New Orleans, Louisiana No. 2017-1117	Deposition (Chemours Company)

Date	Case Name	Case Number	Deposition or Trial
3-Oct-2018	Rosalind Henry and Frederick C. Henry v. Brenntag North America, et al. (Motley Rice LLC; W. Christopher Swett, Esq.)	Superior Court of New Jersey, Middlesex County No. MID-L-1748-17AS	Trial (Johnson & Johnson, Johnson & Johnson Consumer Inc., and Imerys Talc America, Inc.)
17-Oct-2018	Carol Kerkhof, et al. v. Brenntag North American, INC, et al. (Simon Greenstone Panatier Bartlett, PC)	Circuit Court for Montgomery County No. 439392-V	Depositon (Johnson & Johnson, Johnson & Johnson Consumer Inc., and Imerys Talc America, Inc.)
26-Oct-2018	Anastasia Brower, a minor, through her legal guardian Pamela Russell, and Pamela Russell, as the executrix of the Estate of Diane Brower, deceased v. Johnson & Johnson, et al.	In the State Court of Fulton County Fulton State of Georgia No. 16-EV-005534-E	Deposition (Johnson & Johnson, Johnson & Johnson Consumer Inc.)
9-Nov-2018	Paul E. Beach and Rheta E. Beach, Pltfs. vs. 3M Company, etc., et al.	Superior Court of the State of California, County of Alameda - Court of Unlimited Jurisdiction. Case No. RG18893273	Deposition (Rockwell Automation)
13-Dec-2018	Terry Lee Siegfried v. 3M Company, etc., et al. (The Lanier Law Firm; Mark A. Linder, Esq.)	Los Angeles County- Superior Court- Case No. BC691900	Deposition (Rockwell Automation)
9-Jan-2019	Joseph Woon-Shing Lee and Marina Lai-Kuen Lee vs. A. W. Chesterton Company, et al. (Shingler Law; Ronald J. Shingler)	Solano County - Superior Court - Fairfield, CA Case # FCS050176	Deposition (Johnson & Johnson, Johnson & Johnson Consumer Inc.)
25-Jan-2019	Phillip Luna v. The Kerry Group, Inc. et al. (TORHOERMAN LAW, LLC)	Los Angeles County- Superior Court- Case No. BC544985	Deposition (PENTA, et al.)
22-Feb-2019	Lester D. Gardner and Marilyn A. Gardner, etc. vs. ABB INC., etc., et al. (Weinstein Couture, PLLC; Brian D. Weinstein)	Pierce County - Superior Court - Olympia, WA Case No. 172112033	Deposition (Rockwell Automation)